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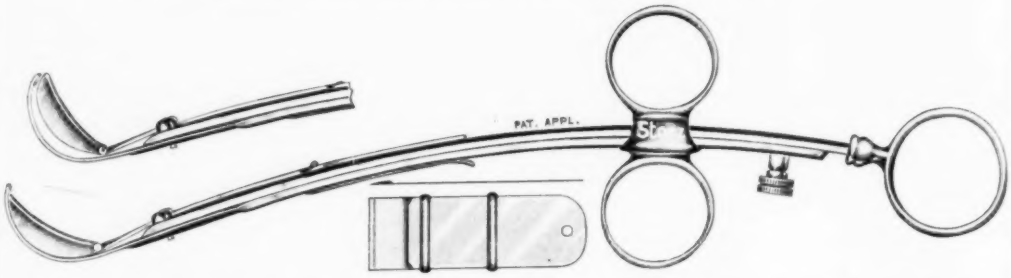

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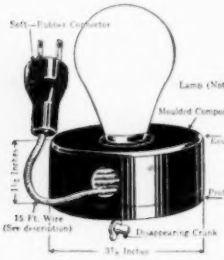

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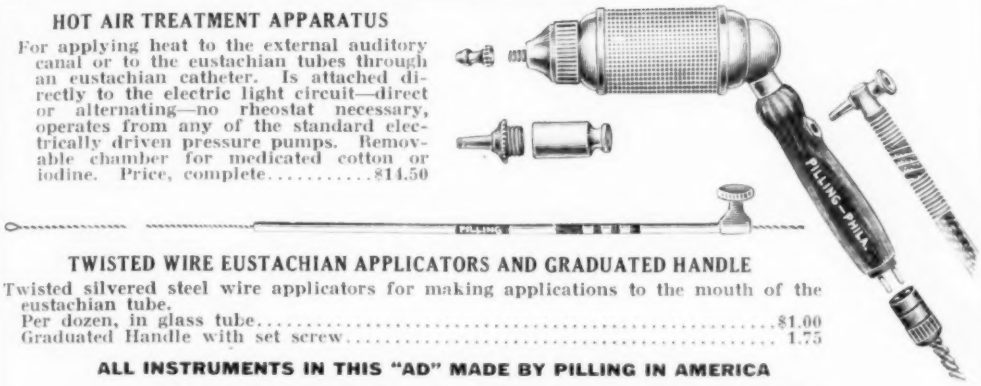

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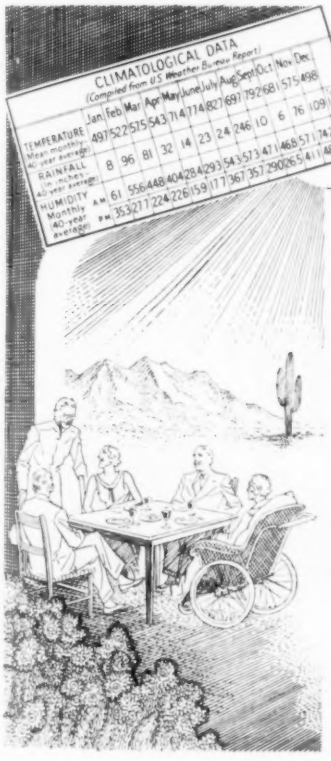

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PULMONARY SYMPTOMS DUE TO ESOPHAGEAL DISEASE

CHEVALIER JACKSON, M.D.

AND

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PHILADELPHIA

So many patients with pulmonary symptoms due to overlooked esophageal disease have come to the clinic that we believe attention should be called to this not uncommon and not always recognized clinical association, especially because in many cases lives can be saved by early diagnosis, prophylaxis and treatment.

There are nine ways and means by which pulmonary symptoms may be developed by pathologic conditions in the esophagus and hypopharynx; it is necessary for accurate diagnosis that distinctions be made:

1. Inspiration of infected food, blood or secretions (oral, pharyngeal, nasal), which overflow into the larynx because they cannot get down a stenosed esophagus.

2. Direct extension upward and over the laryngeal rim by pathologic processes originating in the esophageal or hypopharyngeal wall.

3. Direct extension through the esophagotracheal party wall or through the wall of a bronchus, usually the left main bronchus.

4. Direct extension of esophageal disease to the pleura or through the pleura into the parenchyma of the lung.

5. Direct extension of an esophageal lesion to the mediastinum, thence to the lung.

6. Extension of esophageal disease by way of the blood vessels or lymph channels.

7. Compressive stenosis of the trachea or bronchus without pathologic involvement of the tracheobronchial wall, by bulky esophageal lesions or esophageally lodged, large foreign bodies.

8. Laryngeal paralysis by pressure or involvement of the recurrent nerve by a lesion such as a carcinoma.

9. Reflex symptoms, especially cough, excited by disease limited to the esophagus.

Read before the Section on Laryngology, Otology and Rhinology at the Eighty-Fourth Annual Session of the American Medical Association, Milwaukee, June 16, 1933.

INSPIRATION OF INFECTED MATERIALS

Undoubtedly, at night a small amount of secretion overflows into the larynx of every one who does not sleep prone without a pillow; but with an active cough reflex (the watch-dog of the lungs) this intruding secretion is promptly expelled before it has had time to reach the deeper air passages. With normal unobstructed esophageal drainage, however, the amount of overflow is relatively small. Physiologists estimate that about 1,250 cc. of saliva is secreted daily. Of this, perhaps less than half trickles unconsciously down the esophagus into the stomach independent of the taking of food. The sight of food increases the flow, and the taking of food greatly increases the amount. A little of the downflow is by capillarity through the cricopharyngeal pinchcock, but most of it is by voluntary or subconscious swallowing movements. When as the result of total atresia of the esophagus the saliva cannot reach the stomach, two results have been noted by us: (1) The total amount of saliva secreted gradually diminishes in total quantity, drooling becoming less troublesome, and (2) nutrition, especially in children, suffers for want of some element that saliva normally supplies.[1] This failure of nutrition probably lowers the child's resistance to the onset of pulmonary complications as well as to acute infective pulmonary diseases. The chief active factor in these cases, however, is the inspiration into the tracheobronchial tree of the saliva laden with the pathogenic organisms that lurk in the mouth, about the teeth, in the drainage from the nasal sinuses and in the lymphoid tissue.[2] That the incidence and mortality of pulmonary diseases due to invasion by this route are no greater is evidence of the defensive power of the lungs against infections by the way of the tracheobronchial mucosa.[3] The patient, even though he be a physician, is seldom aware of the fact that the secretions coughed out have been previously inspirated; the subjective localization is pulmonary.

The following is a list of esophageal and compressive periesophageal abnormal conditions we have found causing pulmonary symptoms by one or more of the ways and means mentioned: congenital anomalies;

1. Jackson, Chevalier: Saliva in Nutritive Processes: Clinical Observations in Cases of Cicatricial Atresia of the Esophagus, Arch. Pediat. **40**:326 (May) 1923.

2. Jackson, Chevalier: Peroral Endoscopy and Laryngeal Surgery, St. Louis, Laryngoscope Co., 1915. (This book is out of print, but is in most libraries. The French translation, entitled, "Endoscopie et chirurgie du larynx," is still obtainable from the publisher, Gaston Doin, Paris.)

3. Jackson, Chevalier: Suppurative Diseases of the Lung Due to Inspirated Foreign Body Contrasted with Those of Other Etiology, Surg., Gynec. & Obst. **42**:305 (March) 1926. Jackson, Chevalier, and Jackson, Chevalier L.: Bronchoscopical Observations on Post-Operative Pulmonary Complications, Ann. Surg. **97**:516 (April) 1933.

trauma; foreign body; acute esophagitis; cicatricial stenosis; preventriculosis (so-called cardiospasm); diverticulum, pharyngeal and esophageal aneurysm; actinomycosis; tuberculosis; syphilis; varices; paralysis of the constrictors, tongue, palate, larynx and esophagus; benign growths; malignant growths; glandular masses and periesophageal abscess.

Trauma.—The most frequent trauma to the esophagus is the perforation of the esophageal wall by a blindly passed instrument such as a bougie. A feeding tube pushed down with a stylet has inflicted fatal trauma; even a stomach tube can under certain circumstances cause such a perforation.[4] If the pleural cavity is entered, pneumothorax, pleural shock and septic pleuritis immediately follow. Prostration, pallor, pyrexia and pain in the back and on the involved side, usually increased on deep breathing and by coughing, are the chief symptoms. These cases of trauma are becoming less frequent as the dangers of blind instrumentation are becoming generally realized. An impacted foreign body such as an artificial denture or a pointed object like a safety-pin or bone may cause trauma with pulmonary symptoms.

Foreign Body.—There are five ways in which pulmonary symptoms may be caused by a foreign body in the esophagus.

1. The normal salivary drainage containing all the infective organisms from the nasal accessory sinuses, the tonsils and the teeth may be inspirated into the tracheobronchial tree. The presence of a large obstructive foreign body in the esophagus prevents this drainage from escaping into the stomach as it constantly and normally does. The presence of particles of vegetal foods, by the reaction they create in the tracheobronchial tree, will alone produce suppuration in the bronchial mucosa.[2]

2. Compression stenosis of the trachea may cause dyspnea and a stridorous or wheezing respiration with cough, closely simulating in some cases an asthmatic condition, and in others tracheobronchial diphtheria, influenza and other conditions. Our records show many cases in which a foreign body in the esophagus has led to an erroneous diagnosis of asthma, diphtheria and whooping cough.

3. The inflammatory zone surrounding an irritating foreign body has in a number of instances extended to the upper orifice of the larynx and thence into the endolaryngeal tissues, resulting in edema of the glottic margins and subglottic areolar tissue and simulating closely an acute, infective laryngotracheitis; in some instances this process has been mistaken for laryngeal diphtheria and in other cases for influenza.

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4. Jackson, Chevalier: Carcinoma and Sarcoma of the Esophagus: A Plea for Early Diagnosis, Am. J. M. Sc. **169**:625 (May) 1925.

4. Esophagotracheal fistula from the ulcerative processes resulting from the prolonged sojourn of an overlooked foreign body in the esophagus [5] has occurred in a number of patients coming to our clinic. The resulting pathologic process included suppuration, tracheobronchitis, pneumonitis, solidification with multiple abscesses and empyema, with all the symptoms usually associated with these lesions.[5a] The right lung showed greater involvement.

In a number of our cases, some of which have been reported,[6] an esophagobronchial fistula resulted from ulceration by a foreign body in the esophagus into the left bronchus. At the crossing of the left bronchus, the bronchial and esophageal walls are almost in contact. The same lesions were noted as in the cases with tracheal fistula, but in these bronchial cases the left lung was the one involved. In one case the empyema discharged into the subcutaneous cellular tissue.[5a]

REFLEX PULMONARY SYMPTOMS IN ESOPHAGEAL DISEASE

The intimate anatomic and physiologic association between the sympathetic and pneumogastric nervous systems renders it logical to assume that a reflex cough may derive its excitative impulses from disease of the esophagus. This hypothesis is so seductive, however, that one must be on one's guard to avoid the error that has been made only too often in resting on a diagnosis of "reflex cough" without proper search for organic disease.

Tussive Excitation Center in the Lower End of the Esophagus.—For many years we have noted that many patients cough when the distal end of the esophagoscope approaches the diaphragmatic pinchcock. In some of these patients there was a chronic nonproductive cough; others had chronic suppurative bronchitis, but in still others there was no cough except when excited by the esophagoscopic contact. The free and intricate anastomoses of the sympathetic system render the exact pathway of the afferent impulses uncertain and difficult to determine, but there is no question as to the clinical fact that cough is often present in patients with lesions at the lower end of the esophagus, such as peptic ulcer, static esophagitis, preventriculosis, varices, Banti's dis-

5. (*a*) Jackson, Chevalier: Chronic Nonspecific Infections of the Lungs, J. A. M. A. **87**:729 (Sept. 4) 1926. (*b*) Jackson, Chevalier, and Jackson, Chevalier L.: Les maladies de l'œsophage, Paris, Presses Universitaires de France, 1932.

6. Jackson, Chevalier, and Coates, George M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929. Jackson, Chevalier, and Jackson, Chevalier L.: Surgery of the Larynx, Trachea and Bronchi, in Lewis, Dean: Practice of Surgery, Hagerstown, Md., W. F. Prior Company, Inc., 1930, vol. 4, chap. 6. Jackson.[5a]

ease and carcinoma. We have noted it also in cases of foreign body in this region.

Preventriculosis (So-Called Cardiospasm).—Many of the patients with this disease have a chronic cough, usually productive. Many of them complain of being wakened in the night by accumulated secretion requiring cough for expulsion; sometimes the sputum contains particles of food, and sometimes it has the taste of stale food. Clinical evidence indicates that at least two factors are concerned, namely: (1) inspiration of the overflowing stagnant infected food from the dilated esophageal reservoir and (2) excitation of cough by the chronic static esophagitis at the lower end of the esophagus, as mentioned in a previous paragraph.

CASE 1.—*Chronic Suppurative Bronchitis and Incipient Bronchiectasis Secondary to Preventriculosis.*—A man, aged 42, complained of an accumulation of food which would not go through into the stomach. He said that he had no difficulty in swallowing but that the food did "not seem to go down far enough." Occasionally some of it, tasting sour, stale and cheesy, came back up and was spat out. The symptoms referable to the function of swallowing had been present for six or eight years. After the onset of the trouble in getting food through into the stomach, a cough began, which soon was associated with purulent expectoration. Occasionally, particles of food were noted in the sputum. Two or three times during the night the patient was awakened by choking and strangling which required expectoration of secretions, tainted with sour food, for relief.

Esophagoscopy showed the changes typical of preventriculosis, with accumulation of stale food, white, pasty mucosa and a dilated esophagus with a large mass of folds in the lower portion of the dilatation just above the diaphragmatic pinchcock. As soon as this region was reached by the mouth of the tube, the patient began to cough and he coughed constantly as long as the search of these folds continued. On withdrawal of the esophagoscope for 6 or 8 cm., the cough would cease; it would reappear as soon as the mouth of the tube was pushed down, and the search of the folds in the region of the diaphragmatic pinchcock was resumed. On physical examination, a great variety of râles were found with a few isolated areas of impaired percussion note and hyperresonance. Roentgen examination showed rather heavy peribronchial shadows with increased density in the lower lobe of the right lung. An opaque mixture showed a moderately dilated thoracic esophagus with a persistently closed diaphragmatic pinchcock.

Bronchoscopy showed the appearances typical of a diffuse bilateral chronic bronchitis, worse on the right side, with beginning dilatation of the bronchi. The mucosa was inflammatory; the spurs were thickened; pus was brought up into the larger bronchi by the tussive squeeze after each time it was aspirated. Bacteriologic examination of the pus showed large numbers of a variety of pyogenic organisms. Hydrostatic dilatation of the diaphragmatic pinchcock over a period of three months resulted in a symptomatic cure of the preventriculosis. As the esophageal stasis improved the cough and expectoration gradually diminished; at the end of the three months the pulmonary symptoms had also disappeared. At the last esophagoscopy the mucosa was normal; the dilatation remained but there was no stasis. Cough was excited by the distal mouth of the tube each time the pinchcock was approached.

Comment.—If this were an isolated case one would hesitate to draw conclusions; but as a clinical fact it is typical of many cases in which the same disappearance of the pulmonary symptoms occurred coincidentally with the disappearance of the stasis of food due to failure of the diaphragmatic pinchcock to relax. It seems probable that in this case the pulmonary disease was secondary to the inspiration of the stagnant secretions that overflowed into the larynx, and that the chronic cough was in part a productive requirement and in part a reflex having its source in the static esophagitis in the lower third.

Cicatricial Stenosis.—The children under our care for cicatricial stenosis of the esophagus are very prone to pulmonary diseases, especially bronchopneumonia. The few patients we have lost have died of this disease. When epidemics of acute infective laryngotracheitis are prevalent, the children with esophageal stenosis suffer most severely. The children admitted with total atresia of the esophagus almost all have a loose cough, with suppurative tracheobronchitis. They waken at night choking and strangling with the saliva that has accumulated and overflowed into the larynx. The night nurses are instructed to keep the child face downward without a pillow as much as possible. When the child becomes restless he is turned to the other side, but the mouth is again turned downward to allow the saliva to flow out. Under this treatment chronic bronchial suppuration usually subsides.[1] Of course, deficiencies in vitamins are prevented by routine measures in these cases.

Esophageal Anomalies.—As is well known, infants with congenital malformations of the esophagus usually die of bronchopneumonia, because of the intercommunication between the trachea and the esophagus. It is this, usually, that defeats the attempt to save life by gastrostomy or other procedures. The lower segment of the esophagus communicates with the trachea, and if the upper segment of the esophagus does not anomalously communicate also, the accumulating secretions in the upper segment overflow and pour into the larynx whence they are inspirated into the lungs. Congenital stenosis of the esophagus without communication is also associated with bronchitis from overflow of secretion. It is usually a condition curable by dilation. The following case is illustrative.

CASE 2.—*Chronic Purulent Bronchitis Due to Congenital Stenosis of the Esophagus.*—A girl, aged 17 months, could not take solid or soft solid food. It had been noticed that since birth she had had trouble, "vomiting" (regurgitation) unless liquid food from the bottle was taken very slowly. A special nipple with one minute aperture had been necessary to make the swallowing slow enough. For a day or two at a time, on a number of occasions, nothing would go down. The child would start up suddenly in the night with a choking, coughing, strangling paroxysm. She had four attacks of bronchopneumonia with incomplete

recoveries between them. The last attack had been about two months before admission; a loose cough remained. The temperature ranged to 100 F. every day. It was because of the pulmonary symptoms that the child had been sent to the clinic for diagnostic bronchoscopy. The physical signs were strongly suggestive of chronic bronchitis.

Roentgen examination showed diffuse chronic pneumonitis. Because of the history of difficulty in swallowing since birth, an esophagoscopy was deemed indicated. Before this was done the child was sent to the roentgenologic department for esophageal examination with an opaque mixture. It revealed a smoothly bordered stricture of very small lumen in the middle third of the thoracic esophagus. At esophagoscopy the esophagus was found dilated; the dilated part was just large enough to admit a no. 8 esophagoscopic filiform sound. The walls of the funnel leading to this opening were smooth and free from cicatrices. The mucosa was slightly inflammatory but not ulcerated or eroded. At diagnostic bronchoscopy the tracheobronchial tree was found to contain a large amount of pus. On the aspiration of the pus, the mucosa was reddened, and there were areas of bleeding erosion. The pus showed a variety of pyogenic organisms.

Within three months esophagoscopic dilation resulted in a good lumen ample for swallowing, and after six months the esophagus was apparently of normal size. After three bronchoscopic aspirations in the first two weeks, the defensive power of the lung seemed to be restored; no more bronchoscopic aspirations were done. For about three weeks the child was kept face downward without a pillow. After that time the esophageal drainage was so good that this postural drainage of saliva was unnecessary. Recovery was complete as to both the esophageal and the pulmonary condition. At the end of two years the child, then 3½ years old, was in perfect health. There had been no recurrence of either the difficulty in swallowing or the pulmonary symptoms.

Comment.—The very clear history, the esophagoscopic findings, the roentgen observations and the results of treatment seem to justify the opinion that this was a case of recurrent bronchopneumonia with, in the intervals, bronchial suppuration due to overflow of food and secretions that could not get down the esophagus through a congenital stenosis. The yielding of the latter to peroral esophagoscopic treatment is quite usual in cases of congenital stricture when there is no communication between the esophagus and the trachea or bronchus. When a child has bronchial trouble, it would seem advisable to have a roentgen examination of the function of swallowing in addition to examination of the chest.

PARALYSIS OF THE PHARYNX AND ESOPHAGUS

Pulmonary symptoms from overflow may occur in cases of impaired or lost motility of the tongue, palate, larynx, constrictors or peristaltic muscular coat of the esophagus; one or more of these paralyses may be combined. The esophageal drainage depends largely on swallowing movements, mostly subconscious; the aid of gravity is slight; a normal person can swallow up-hill easily, as in kneeling at a spring, but he cannot swallow when erect if the constrictors and peristaltic muscu-

latures are paralyzed. Inspiration of the overflowing oral secretions in these cases quickly results in pulmonary complications, as in myasthenia gravis and bulbar conditions generally.

SYPHILIS OF THE ESOPHAGUS

Syphilitic disease of the esophagus may produce pulmonary symptoms in any of the ways indicated. The following case is illustrative of the necessity for diagnostic study of the esophagus in cases of suppurative bronchitis.

CASE 3.—*Suppurative Bronchitis from Inspiration of Overflow in a Case of Syphilis of the Esophagus.*—A man, aged 47, was referred for bronchoscopic aspiration of an abscess of the lower lobe of the right lung following bronchopneumonia. By questioning there was elicited a history of sudden attacks of choking and strangling with secretions at night preceding the bronchopneumonia. The sputum was copious and malodorous. Movements of the tongue and palate were slightly impaired.

Roentgen examination showed a diffuse inflammatory process throughout both lungs, worse on the right, but nothing localized that could be called an abscess. The aortic shadow was broader than normal. The roentgen diagnosis was aneurysm of the aorta plus bronchiectasis. Mirror examination revealed a left recurrent paralysis and a puddling of secretion in both pyriform sinuses. The serologic test was positive (+ 4), and as roentgen examination showed a dilated aorta, a tentative diagnosis of recurrent paralysis due to aneurysm was made. Because of the puddling of secretions in the pyriform sinuses esophagoscopy was advised, and the patient was sent to the roentgenologist for an examination of the esophagus with an opaque mixture. The report was as follows: "narrowing of the esophagus at the level of the dilated aortic arch; as the border of the shadow is irregular we are inclined to believe that it is a malignant infiltration. An esophagoscopic examination could determine between these two possibilities, if the probable presence of an aneurysm is not deemed a contraindication to endoscopy."

The internist called attention to the possibility of bulbar trouble. He could find no clinical evidence of aneurysm and advised esophagoscopy to clarify the diagnosis. Esophagoscopy revealed a rather flat, ulcerated, bleeding lesion without fungations in the upper third of the thoracic esophagus. The esophagoscopic diagnosis was: "Stenotic ulcerative esophagitis, the character of the ulcer to be determined by biopsy; specimens of exudate taken with the mechanical spoon and a specimen of tissue with the biting forceps."

The histologic and bacteriologic reports were as follows: There was no evidence of pale Spirillum in the scrapings or of malignancy in the tissue submitted. The process was one of chronic ulcerative inflammation with cellular changes strongly suggestive of broken-down gumma. Bronchoscopy showed a chronic suppurative mucosal inflammation limited to the larger bronchi. The mucosa of the smaller bronchi seemed relatively normal. About 20 cc. of thick pus was aspirated. Microscopically, it was found to contain a number of starch granules and a mixture of bacteria and fungi.

Under energetic antisyphilitic treatment, including mercurial inunctions and potassium iodide internally, the puddling of secretions in the pyriform sinuses disappeared, the nocturnal attacks of choking and strangling, the cough and the expectoration ceased, and the swallowing, which was not really difficult at any

time, became normal. Roentgen examination of the function of swallowing showed nothing abnormal. At esophagoscopy a small scar was found at the site of the former ulcer; the esophagoscope (9 mm. by 53 mm.) passed all the way to the stomach without encountering stenosis. Bronchoscopy showed the tracheobronchial tree free from pus and the mucosa normal. The paralyzed vocal cord did not recover motility, but the voice had not been much impaired and remained quite good. The tentative diagnosis of aneurysm was abandoned as unsupported by further studies, especially the esophagoscopic findings.

Comment.—This interesting case was referred to the bronchoscopic clinic for aspiration of a pulmonary abscess. Puddling of secretion and a recurrent paralysis noted on mirror examination led to a suspicion of esophageal disease as the cause of the pulmonary symptoms, in the absence of any definite complaint of difficulty in swallowing. Esophagoscopic biopsy resulted in the diagnosis of syphilitic ulcer of the esophagus; this was later confirmed by the progress of the case under antisyphilitic treatment.

ANEURYSM

Aneurysm may cause respiratory symptoms in two ways unmentioned in the textbooks: (1) Compressive stenosis or erosive invasion of the esophagus may lead to the overflow and inspiration of secretion, or (2) bilateral paralysis of the recurrent nerve, by an increase in size, may produce asphyxia, stridor, wheezing, loose cough, all kinds of râles, drowning of the patient in his own secretions,[7] bronchopneumonia and chronic suppurative disease of the trachea, bronchi and lungs. The secretions cannot be expelled because of impaired glottic cooperation in the tussive cycle.

MYCOTIC STENOSIS OF THE ESOPHAGUS

The following case is an interesting example of mycotic disease of the esophagus with pulmonary complications.

CASE 4.—*Actinomycosis of the Lung Secondary to Actinomycosis of the Esophagus.*—A man, aged 44, complained of progressively increasing difficulty in swallowing; it had reached the point where even liquids were taken with difficulty and the saliva had to be spat out frequently. Sometimes the patient would awaken at night with a choking sensation. There was no cough or other symptoms.

Physical examination of the chest revealed no abnormality. Roentgen examination of the heart and lungs showed no abnormality, but the use of an opaque mixture revealed a smoothly bordered shadow of esophageal stricture with a minimum lumen of about 3 mm. for a vertical distance of about 4 cm. Esophagoscopy showed the stenosis to be firm, annular and concentric. The mucosa in the strictured area was whitish, granular and eroded. Histologically and bacteriologically, a specimen showed the lesion to be actinomycosis.

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7. Jackson, Chevalier: Drowning of the Patient in His Own Secretion, Laryngoscope **21**:1183 (Dec.) 1911.

The stenosis was dilated, and the patient was given potassium iodide internally, during a period of three months. At the end of this time an 8 mm. esophagoscope (externally 9 mm. by 11 cm.) would pass through the stenosis; the mucosal surface was improved but not altogether of normal appearance. The swallowing was subjectively normal.

The patient neglected to return until two years later, when he came not for dysphagia but because of cough, profuse expectoration and loss of weight. Esophagoscopy showed about the same mucosal condition as when he was last seen; the esophageal lumen through the narrowed portion was large enough to permit the passage of an 8 mm. standard esophagoscope. All sorts of râles were audible over both lungs. Percussion elicited an impaired note in small areas at various locations in both lungs. The roentgenologic report was as follows: "a widespread diffuse inflammatory process over both lung fields, suggestive of early extensive tuberculosis. No cavities with a fluid level can be made out, but in both lower lobes there are areas rather suggestive of beginning cavitation." The sputum was negative for tuberculosis.

Bronchoscopic examination revealed large quantities of pus. When the pus was aspirated the tracheobronchial mucosa was found to be inflammatory, with patches of reddish and whitish areas that bled when touched. Specimens of whitish material were removed with the mechanical spoon, and a specimen was taken from a ridgelike elevation on the wall of the bronchus of the right lower lobe. The tussive squeeze brought up small quantities of pus from various small branches in both lungs. Histologic examination of the specimen of tissue showed only chronic inflammatory changes without a specific character. The residual pus and the scrapings, however, were permeated with actinomycotic organisms. The patient returned home and was reported to have died about six months later of pulmonary abscesses.

Comment.—The advanced esophageal actinomycosis with apparently normal lungs at the time the patient was first seen would seem to justify the inference that the pulmonary lesion was secondary, and it seems logical to conclude that inspiration of the overflowing secretions carried the infection into the lungs.

BENIGN AND MALIGNANT GROWTHS OF THE ESOPHAGUS

Any new growth of the esophagus, if obstructive, may cause pulmonary complications or symptoms from the overflow and inspiration of oral secretions. Additionally, malignant growths may cause such symptoms by (1) overflow, (2) direct invasion of the air passages or pleura, (3) metastases, most often glandular, or (4) paralysis of the recurrent nerve, and (5) cough may be excited reflexly by irritation of the tussive center in the lower end of the esophagus referred to in a preceding paragraph. Benign growths of the esophagus are rare; malignant growths, unfortunately, are common. Cough is a common and often an early symptom of carcinoma of the esophagus,[4] and this condition should always be thought of as a diagnostic possibility in cases of unexplained cough. Numerous examples of all the foregoing conditions are shown in our case records.

PROPHYLAXIS

Prophylaxis is of the utmost importance. In every case of obstructive esophageal disease the risk of pulmonary complications should be borne in mind and precautions taken to prevent overflow and inspiration of secretions into the air passages.

Buccal Drainage by Posture.—During sleep a pillow should be dispensed with, and the face of the patient should be turned downward toward the bed on which is placed a pad of absorbent gauze. When the patient becomes restless, he can be turned on the other side; he should not, however, sleep on his arm. In this position saliva and to some extent nasal and nasopharyngeal secretions will find their way out through the mouth. In the patient's waking hours he should expectorate secretions that cannot be swallowed. In the care of patients after laryngectomy, diverticulectomy, fulguration, diathermy and other procedures involving the pharynx, it may be impossible or undesirable for the patient to swallow. In such cases, as a precaution, continuous aspiration with a "mouth-hook" tube must be resorted to if the particular operation renders the aforementioned posture objectionable in the after-care. Prevention of chronic suppurative bronchitis and the bronchiectatic septic tank depends on the early diagnosis and treatment of the underlying esophageal condition and especially on prevention of stagnation by regular bronchoscopic aspirations.

Restoration of Esophageal Drainage.—The next important indication is to restore esophageal patulency as soon as possible by appropriate treatment.

TREATMENT

Elimination of the etiologic factors is the chief indication; so long as they persist, only prophylaxis is possible. Esophageal drainage must be reestablished as soon as may be feasible. Meanwhile the aforementioned prophylactic measures must be carried out. Treatment of the secondary bronchitis, the bronchopneumonia or other pulmonary lesions is chiefly by bronchoscopic aspiration and the avoidance of drugs that inhibit the cough reflex or those that lower bechic efficiency by increasing viscosity.[8]

8. Jackson, Chevalier: Postulates on the Cough Reflex in Some of Its Medical and Surgical Phases, Therap. Gaz. **44**:609 (Sept.) 1920; Cough: Bronchoscopic Observations on the Cough Reflex, J. A. M. A. **79**:1399 (Oct. 21) 1922; footnote 2. Jackson, Chevalier, and Jackson, Chevalier L.: Peroral Pulmonary Drainage, Tr. Am. Therap. Soc., 1933, to be published. Jackson, Chevalier: Endoscopic Treatment of Suppurative Diseases of the Bronchi and Lungs, Siglo méd. **90**:565 (Nov. 26) 1932; Endoscopic Treatment of Suppurative Diseases of the Bronchi and Lungs: Postulates for Discussion, résumé supplementary to a report on the same subject at the Second International Congress of Otorhinolaryngology, Madrid, Comptes Rendus, Fasc. premier, September, 1932, p. 1; abstr., Arch. Otolaryng. **18**:399 (Sept.) 1933.

CONCLUSIONS

1. The frequent overlooking of the esophageal origin of pulmonary symptoms has led us to analyze the clinical data at our clinic to render it available for diagnostic purposes.

2. There are nine ways and means by which pulmonary symptoms may be developed by pathologic conditions in the esophagus and hypopharynx; it is necessary for accurate diagnosis that distinctions be made: (*a*) inspiration of infected food, blood or secretions (oral, pharyngeal, nasal), which overflow into the larynx because they cannot get down a stenosed esophagus; (*b*) direct extension upward and over the laryngeal rim by pathologic processes originating in the esophageal or hypopharyngeal wall; (*c*) direct extension through the esophagotracheal party wall, or through the wall of a bronchus, usually the left main bronchus; (*d*) direct extension of esophageal disease to the pleura or through the pleura into the parenchyma of the lung; (*e*) direct extension of an esophageal lesion to the mediastinum and thence to the lung; (*f*) extension of esophageal disease by way of the blood vessels or lymph channels; (*g*) compressive stenosis of the trachea or bronchus without pathologic involvement of the tracheobronchial wall, by bulky esophageal lesions, glandular metastases or esophageally lodged large foreign bodies; (*h*) laryngeal paralysis by pressure or involvement of the recurrent nerve by a lesion such as a carcinoma; (*i*) reflex symptoms, especially cough, excited by disease limited to the esophagus.

3. The pulmonary symptoms in some cases predominate over the causative esophageal symptoms so strongly that the latter may be entirely overlooked by the patient and physician alike.

4. The characteristic pulmonary symptom of esophageal disease most often found is sudden wakening with coughing, choking and strangling. The source of the cough is, subjectively, altogether laryngotracheal, and the patient does not realize that the accumulated secretions have previously overflowed into the larynx. To a slight extent this overflow, as well as this symptom, may occur in health, but it is much more prominent in cases of impaired esophageal drainage from even slight degrees of stenosis.

5. Puddling of secretions in the pyriform sinus as seen in the laryngeal mirror is an important, early, subjective sign of esophageal stenosis with potentially impending pulmonary complications.

6. The following is a list of esophageal and compressive, periesophageal, abnormal conditions we have found causing pulmonary symptoms by one or more of the ways mentioned: congenital anomalies; trauma; foreign body; acute esophagitis; cicatricial stenosis; preventriculosis (so-called cardiospasm); diverticulum, pharyngeal and esophageal; aneurysm; actinomycosis; tuberculosis; syphilis; varices;

paralysis of the constrictors, tongue, palate, larynx and esophagus; benign growths; malignant growths; glandular masses, and peri-esophageal abscess.

7. In the search for obscure causes of cough esophageal examination with the roentgen-rays and the esophagoscope should not be overlooked.

ABSTRACT OF DISCUSSION

DR. GABRIEL TUCKER, Philadelphia: I appreciate the privilege of discussing the Jacksons' presentation. This is like all their papers—when they are through, there is nothing to add, and there can only be commendation for the material presented and the manner of presentation. I wish to mention three cases in which there were both pulmonary and esophageal disease, and in which the diagnosis of the true condition was made by direct examination.

The first case is that of a child, aged 12 months, who presented signs of obstructive dyspnea, wheeze, stridor and inspiratory indrawing at the suprasternal notch and the tip of the ensiform. There was a history of the child choking while sucking a chicken bone. The child swallowed liquids readily. Roentgen examination of the esophagus, both with and without an opaque mixture, showed no evidence of an esophageal foreign body.

Bronchoscopy was done, and the larynx and tracheobronchial tree were found to be normal, except for a bulging forward of the wall of the esophagus just above the bifurcation, producing a compression stenosis of the trachea. On esophagoscopy, the upper epiphyseal end of the leg bone of a chicken was found in the esophagus. A notch in the bone allowed liquids to pass without difficulty. After esophagoscopic removal of the foreign body, the tracheal compression disappeared.

The second case is that of a boy, aged 13, who was admitted to the Phipps' Institute of the University of Pennsylvania because of a chronic productive cough, loss of weight and general malaise. Roentgen examination by Dr. McPhedran showed an extensive lesion involving the upper lobe and hilar region of the right lung. Dr. McPhedran detected, however, what appeared to be a cavity with a fluid level in the region of the suprasternal notch in front of the spine, and when an opaque mixture was given, an enormous dilatation of the entire esophagus was found—preventriculosis or so-called cardiospasm. Esophagoscopy confirmed this finding, and on bronchoscopic examination the pulmonary lesion was found to be a chronic purulent tracheobronchitis with pneumonitis of the right lung. Repeated examination of the secretions from the lung failed to show acid-fast organisms. The hiatal esophagus was dilated by passing an esophagoscope, and after several dilations swallowing was much improved. Bronchoscopy was repeated, and with improvement in swallowing, the patient no longer aspirated the overflow secretions from the esophagus and the pulmonary lesion rapidly cleared up. The boy has gained weight and has shown marked general improvement.

The third case was that of a man, aged 56, who complained of progressive loss in weight, with increasing difficulty in swallowing over a period of six months. The picture and history were typical of cancer of the esophagus. There had been no complaint of cough or hemoptysis. Roentgen examination with an opaque mixture showed a long, smooth narrowing of the lower third of the thoracic part of the esophagus. Increased density in the left hilar region and a small area of atelectasis in the posterior portion of the lower lobe of the left lung were noted.

Esophagoscopy showed an annular compression stenosis of the lower thoracic part of the esophagus with a smooth mucous membrane. No evidence of infiltration or ulceration was found, and without undue pressure an 8 mm. esophagoscope passed into the stomach, dilating the constricted area. The passage of the esophagoscope relieved the difficulty in swallowing.

Because of the roentgen evidence of disease of the left lung, bronchoscopy was performed, and a fungating lesion was found in the main stem of the bronchus of the left lower lobe. Biopsy showed cancer of the bronchus.

Dr. John B. Potts, Omaha: I feel greatly honored to be asked to discuss a paper written by the Jacksons. They always seem to find something new in their particular specialty which is of practical value.

Owing to shortness of time, they did not emphasize the benefit that can be derived from the use of the bronchoscope in cases of bulbar paralysis resulting in paresis of the muscles of deglutition and inspiration of large quantities of fluid.

The reverse symptom which Dr. Tucker mentioned in his last case may occur with a foreign body. I had one case in which a patient had all the symptoms of a foreign body in the esophagus, but on examination it was found to be in the left main bronchus. The foreign body was a short heavy needle used for injecting material into the tonsils for tonsillectomy. I thought probably that the reversed symptoms were due to the weight of the foreign body.

Dr. Samuel Iglauer, Cincinnati: The intimate anatomic relationship of the esophagus and the trachea often leads to pulmonary manifestations in esophageal disease. The classic illustration of this occurs in congenital atresia of the esophagus; in this condition an incomplete cleavage occurs between the esophagus and the trachea, both of which originate from the foregut of the embryo. As a result of the incomplete cleavage, the trachea and the esophagus communicate with each other through a fistula, and the infant usually dies of pneumonia, either from regurgitation of gastric juice or from a spill-over from the blind pouch in the esophagus.

At the other extreme of life, a tracheo-esophageal fistula occurs as the result of perforation of an esophageal carcinoma into the trachea. A tracheo-esophageal fistula can be readily demonstrated by placing a string tampon in the esophagus below the suspected site of the fistula (esophagoscope). The patient then swallows iodized poppy seed oil 40 per cent which may be seen to enter the bronchi.

Malignant disease of the upper end of the esophagus often manifests itself by swelling or edema in the region of the arytenoid cartilage, leading to laryngeal symptoms. An esophageal carcinoma occasionally invades the recurrent laryngeal nerves, producing paralysis of one or both vocal cords. It is therefore important to examine the hypopharynx and the upper end of the esophagus in cases of this kind in order to make a correct diagnosis. In advanced carcinoma of the esophagus, metastases may invade the bronchial glands at the bifurcation of the trachea and give rise to pulmonary symptoms.

In cases of diverticulum of the esophagus, as well as in cardiospasm, the patients frequently complain of a nocturnal cough, and not infrequently are treated for chronic bronchitis. The cough, of course, results from a spill-over of the retained fluids in the esophagus, which trickle into the trachea when the patient is recumbent and asleep.

A foreign body impacted in the esophagus may interfere with breathing either mechanically or as a result of the inflammatory edema occurring about the foreign body and extending to the air passages.

The observation made by Drs. Jackson that a cough reflex can be elicited from the lower end of the esophagus is interesting. The layman often speaks of a "stomach cough," and possibly this cough really originates in the esophagus.

DR. HERMAN J. MOERSCH, Rochester, Minn.: The subject presented has been covered so thoroughly in the paper and by the preceding discussions that there remains little to add. I wish, therefore, merely to show three slides which emphasize the importance of the Jacksons' remarks.

In the first slide is demonstrated a large pulmonary abscess in association with a case of cardiospasm with a markedly dilated sac, the abscess resulting from the overflow from the esophagus into the trachea, especially on lying down and during sleep.

The second slide shows the case of a patient with a large pharyngo-esophageal diverticulum with secondary pulmonary infection due to the overflow of material from the sac into the bronchi. The patient suffered from recurrent attacks of fever and chills which were regarded as attacks of pneumonia, but in reality resulted from pulmonary infection secondary to the diverticulum. Such pulmonary complications in association with a pharyngo-esophageal diverticulum necessarily add to the operative risk in extirpation of the sac.

The last slide demonstrates a case in which there was a fistula between a traction diverticulum of the lower part of the esophagus and the right bronchus of eight years' duration. Owing to the secondary pulmonary involvement, recurrent attacks of fever, chills and bleeding developed, necessitating operation.

It may therefore be seen that disease of the esophagus may give rise to serious pulmonary complications and must be considered whenever operation is to be performed on the esophagus.

DR. M. F. ARBUCKLE, St. Louis: As the laryngeal cartilages in the young child are softer than in an older patient, they permit compression of the larynx by a foreign body in the esophagus with resultant interference with respiration much more readily.

While in infants one of the outstanding symptoms of foreign body in the esophagus is interference with respiration as well as deglutition, in older patients respiratory disturbances are comparatively rare and interference with deglutition may be absent.

I have seen a postmortem specimen of the esophagus and tracheobronchial tree which contained a set of false teeth positively identified as having been lost eighteen years previously. It was lodged in the esophagus and, after having perforated the wall of the esophagus, had also perforated the wall of the bronchus with a resultant fatal bronchopneumonia. I have recently removed a collar button from the esophagus of an infant, aged 3 months, with a history of six weeks' lodgment. The only symptom in this case was interference with respiration. I have removed a jack from the esophagus of a young child whose only symptom was interference with respiration. Three years ago I saw a child, 6 years old, in whom a piece of bone had become lodged in the esophagus while he was eating beef stew. The history was clear as to the time when the foreign body became impacted, but no attempt had been made to have it removed. It finally perforated into the pleural cavity, causing empyema. During treatment of the empyema, an esophageal fistula was discovered; it healed spontaneously.

In my experience one of the outstanding symptoms of diverticulum of the esophagus has been cough, and later interference with deglutition.

In regard to the first case which the Jacksons showed, is it possible that compression of the left main bronchus by a foreign body in the esophagus might have accounted for the occurrence of symptoms confined to the left lung?

DECOMPRESSION OF THE FACIAL NERVE

PHYSIOLOGY OF THE SEVENTH AND NINTH NERVES AND MOVEMENTS OF THE LID IN FACIAL PARALYSIS

EDWARD CECIL SEWALL, M.D.
SAN FRANCISCO

Interest has been aroused in the direct repair of the facial nerve by the work of Bunnell[1] and Martin[2] and by the more recent results of the experimental work of Ballance and Duel.[3] The literature on the subject is well covered in articles written by these investigators. Before reporting two cases of my own in which decompression was done, I shall review the various functions of the interesting seventh nerve. This seems opportune in the light of the recently published work of Reichert and Poth[4] of Stanford University, which makes a distinct contribution to the knowledge of the physiology of the facial and glossopharyngeal nerves.

The motor fibers of the seventh nerve to the facial musculature arise in the pons in a cell column common to all the motor cranial nerves except the hypoglossal nerve and the nerves to the eye. This facial nucleus, included in the basal reflex arc, is controlled from the cortex in a manner that has special clinical significance. While the corticobulbar tract reaches it undivided, there is a separation of the higher centers controlling each nucleus, so that the temporal fibers to the occipitofrontalis, orbicularis oculi and corrugator supercilii are not involved in cortical hemiplegia.

The sensory part of the nerve is not so well understood. While there is general agreement as to the route traversed by the taste fibers,[5] it seems that a new conception must be developed as to the distribution of the secretory fibers since Reichert and Poth's article was published.

From Stanford University School of Medicine.

1. Bunnell, S.: Surg., Gynec. & Obst. **45**:7, 1927.

2. Martin, R. C.: Intratemporal Suture of Facial Nerve, Arch. Otolaryng. **13**:259 (Feb.) 1931.

3. Ballance, C., and Duel, A. B.: Operative Treatment of Facial Palsy by Introduction of Nerve Grafts into Fallopian Canal and by Other Intratemporal Methods, Arch. Otolaryng. **15**:1 (Jan.) 1932.

4. Reichert, F. L., and Poth, E. J.: Proc. Soc. Exper. Biol. & Med. **30**:973, 1933.

5. Lewis, D., and Dandy, W. E.: Course of Nerve Fibers Transmitting Sensation of Taste, Arch. Surg. **21**:249 (Aug.) 1930.

It is believed that the secretory fibers[6] leave the geniculate ganglion and reach the sphenopalatine ganglion by way of the large superficial petrosal nerve. From there the fibers reach the lacrimal gland by way of the maxillary ganglion and its zygomatic terminal.

It has formerly been held that the glossopharyngeal nerve supplies only the parotid gland with its secretory fibers. The consensus is that the fibers of that nerve can be traced through Jacobson's branch via the middle ear and by way of the small superficial petrosal, the otic ganglion and the auriculotemporal nerve to the parotid gland.[6] Reichert and Poth not only question that classic distribution but, apparently, they have proved that the glossopharyngeal nerve furnishes additional distribution to the submaxillary and sublingual glands. Also, they believe that all these fibers must pass through the chorda tympani, as they found secretion from all the homolateral salivary glands diminished on section of the chorda tympani after it left the facial nerve. The observations that led them to devise a new path for the secretory fibers were based on the following evidence:

1. In a case reported by Reichert,[7] relief of tympanic plexus neuralgia followed division of the root of the ninth nerve.

A woman, in 1921, had pain in the left concha. In 1932, she had a sensation of drawing and discomfort in the upper part of the left half of the face. A few months later the sensation involved the cheek, forehead and occipital region. An attack of coryza at that time was followed by a sharp, stabbing pain in the external auditory canal, which caused her to shriek and clutch her ear. Except for an interval of twelve days, she had daily paroxysms of pain deep in the ear. At times there was dull aching in the ear, with burning of the left side of the face following an attack. There was no increase in salivation. Swallowing, eating or talking did not induce paroxysms.

In December, 1932, an operation was performed by unilateral cerebellar approach, with the patient under local anesthesia, and the seventh, eighth, ninth and tenth facial nerves were exposed. Touching the seventh nerve induced pain in the ear which the patient immediately stated was not the tic pain. This sensation was located in the cartilaginous part of the anterior wall of the external auditory meatus. Touching the ninth root caused the patient to shriek; she said that the pain was identical with the tic from which she had suffered for four months. This testing was repeated four times, with similar results. The root of the ninth nerve was then divided. All pain ceased immediately, and the patient fell asleep on the operating table. No pain has occurred since the operation. There has been no anesthesia of the ear or of the canal. Sensation was lost over the soft palate and the pharyngeal wall from the eustachian tube to the epiglottis. Taste was absent in the posterior third of the tongue. No paralysis of the muscles of the soft palate occurred.

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6. Spalteholz, K. W.: Handatlas der Anatomie des Menschen, Leipzig, S. Hirzel, 1895, vol. 3.

7. Reichert, F. L.: Tympanic Plexus Neuralgia, J. A. M. A. **100**:1744 (June 3) 1933.

2. Hesse[8] reported a case in which section of the neck of the glossopharyngeal nerve obviously distal to the branching of Jacobson's nerve resulted in a diminution of parotid secretion.

3. Reichert and Poth observed that secretion from the parotid, submaxillary and sublingual glands was diminished on the homolateral side after section intracranially of the glossopharyngeal nerve.

4. Reichert and Poth observed that secretion from all the salivary glands was reduced on section of the chorda tympani distal to its departure from the fallopian canal.

5. They observed a case of Bell's palsy in which taste was retained, though all homolateral salivary secretion was diminished. This case would indicate that secretory fibers reach the chorda tympani via the facial nerve distal to the chorda tympani.

6. They failed to observe any salivation in a known case of neuralgia of the tympanic plexus.

In accordance with these facts, Reichert and Poth concluded that some secretory fibers leave the salivator nucleus and pass with the seventh motor nerve to the chorda tympani, and that the secretory fibers from the glossopharyngeal nerve join these fibers in the chorda tympani via the anastomosing branch of the ninth nerve in its union with the facial nerve. From the chorda tympani the fibers to the parotid pass through the otic ganglion and the auriculotemporal nerve, while the pathway to the submaxillary and sublingual glands is through the chorda tympani, as heretofore described.

Hunt believed that the fibers of common sensation, which he ascribed to the facial nerve, were distributed about the auricle in the vicinity of the external canal. He[9] attributed the herpetic vesicles occurring in this locality to the fibers of common sensation. He believed that the fibers reached the tympanic plexus in a direction opposite to that taught, and traced them through the large and small superficial petrosal nerves. He believed that otalgia was caused by irritation of these fibers. The case studied by Clark and Taylor,[10] in which pain in the ear was relieved by section of the facial nerve intracranially, apparently substantiated Hunt's conclusions. However, his views have been opposed. The relief of pain has been attributed by his critics to the effects of decompression. They considered that the herpetic manifestations were due to involvement of the fifth nerve or of the neighboring ganglions. Reichert's case apparently proves that tic douloureux of the tympanic plexus is not always due, as Hunt affirmed, to the branches emanating

8. Hesse, E.: Surg., Gynec. & Obst. **52**:114, 1931.

9. Hunt, J. R.: Brain **38**:418, 1915.

10. Clark, L. P., and Taylor, A. S.: True Tic Douloureux of the Sensory Filaments of the Facial Nerve, J. A. M. A. **53**:2144 (Dec. 25) 1909.

from the geniculate ganglion, but to involvement of the glossopharyngeal nerve. The repeated experimental production of the paroxysm in a patient under local anesthesia was effected by irritation of the root of the glossopharyngeal nerve, and prompt and permanent relief of all pain resulted from its section.

Tic douloureux of glossopharyngeal origin was described first in 1910, by Weisenburg. Dandy,[11] in 1927, reported two cases with cure by intracranial section of the ninth nerve, and gathered from the literature reports of eighteen cases. Reichert reported a total of forty-three cases in the literature. In all these cases symptoms other than those in the ear were present. Lancinating pain in the peripheral distribution of the ninth nerve was a constant symptom. The paroxysms were usually induced by swallowing or talking or by other movements of the tongue. Tic douloureux of the tympanic branch of the ninth nerve (Jacobson's nerve) as described and cured by section of the ninth root by Reichert, is characterized by stabbing paroxysmal pain in the ear only and was formerly regarded by Hunt as due to neuralgia of the distribution of the geniculate ganglion. It is a rare neurologic manifestation.

MOVEMENTS OF THE LID IN FACIAL PARALYSIS

Facial palsy, when complete, interferes with the normal movement of the eyelids. A study of normal and abnormal persons has led to some puzzling observations for which I have been able to find no satisfying explanation.

The orbicularis oculi closes the eyelids when the will to close is brought into play. It is innervated by the facial nerve. In complete facial paralysis the eye cannot be wilfully closed. However, if the eye is made to follow vertical movements of the hand, the upper lid will be seen to move upward and also downward past the midline. The downward movement may not result in complete closure of the eye, but it closely approximates the extent of the similarly produced downward movement of the upper lid in a normal person. The upward movement is due to the action of the levator palpebrae innervated by the third nerve. What causes the downward movement? The orbicularis is paralyzed and thus cannot be a factor.

References in the literature to the movements of the lids are meager. Henke and Lubarsch, the American Encyclopedia of Ophthalmology and other authorities are silent as to what force there may be to act in antagonism to the levator in the presence of a completely paralyzed

11. Dandy, W. E.: Glossopharyngeal Neuralgia (Tic Douloureux); Its Diagnosis and Treatment, Arch. Surg. **15**:198 (Aug.) 1927.

orbicularis. One textbook on physiology [12] states briefly that the lowering of the upper lid may be assisted by gravity. This influence is easily shown to be negligible. The effect of gravity can be reversed by everting the patient's head. The lid of the paralyzed eye moves as well in that position.

The fact that the lid moves with the downward turning of the eyeball suggests that it is pulled by cohesion or by the conjunctiva. This may be disproved by the movement of the lid of a person with a glass eye. The lid not only follows what downward movement of the artificial eye there may be, but slides farther downward over the glass in spite of possible deterrent friction. When the head is everted the action remains the same. Some patients with facial paralysis close the eye in sleep, but that is not usual.[13] Wilful attempts to close the eye are unsuccessful and cause the ball to turn upward. Magnus,[13] in 1837, described a patient who closed the paralyzed eye at night, who could not close it wilfully but closed it at the approach of a hand, at the impact of bright light and on sneezing. He suggested that the sympathetic control of the smooth muscles in the lid was relaxed at night. This is in keeping with the narrowing of the palpebral fissure on section of the cervical sympathetic nerve.

Though the muscular form and tone of the orbicularis in a normal person would lead to narrowing of the palpebral fissure in the event of relaxation of the levator palpebrae, it would not explain the extensive downward movement of the upper lid following cortical stimulation. As has been mentioned, this movement occurs in the absence of the eyeball. In the presence of paralysis of the orbicularis this solution becomes untenable.

Various types of palpebral involvement have been studied.

CASE 1.—A patient, seen through the courtesy of Dr. Thomas Inman, with double facial paralysis and muscular atrophy of the Aran-Duchenne type, showed a prompt downward movement of the upper eyelids slightly past the midline on looking downward. Wilful closure of the eyes was not possible.

CASE 2 (referred by Dr. Reichert).—A patient in whom the homolateral sympathetic nerves, including those about the carotid, had been removed, showed marked narrowing of the palpebral fissure and other classic signs of sympathetic paralysis. Upward and downward movements of the lid were normal.

Observations in these cases furnish the following evidence:

1. The upper lid does not move downward on wilful stimulation to close the eye in the presence of complete facial paralysis.

2. The upper lid follows movements of the hand downward past the midline, even in the presence of complete facial paralysis.

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12. Jelliffe, S. E., and White, W. A.: Diseases of the Nervous System, a Textbook of Neurology and Psychiatry, Philadelphia, Lea & Febiger, 1915.

13. Magnus, cited by Hirschfeld: Berl. klin. Wchnschr. **38**:904, 1901.

3. Gravity plays no significant rôle in the downward movement of the lid.

4. Downward movement of the lid induced by downward rotation of the eyeball, because of cohesion to the conjunctiva or because of some other direct influence, has apparently been disproved.

The obvious is often most difficult of explanation. Professor Meyer and Professor Hinsey of the department of anatomy of Stanford University School of Medicine, have given me some interesting suggestions; one called my attention to associated muscular movement, and the other stressed contracture. Certain group relationships are evidenced by the fact that in the wrist drop of peripheral palsy, with paralysis of the common extensors, the wrist cannot be extended except under certain circumstances, although the short extensors are not affected. It is possible to stimulate a muscle electrically, though its nerve is paralyzed. Movement of a paralyzed trapezius muscle has been induced by associated movement of the arm. Although both internal recti are paralyzed, each may function normally in action with the external rectus of the opposite eye in conjugate movements.[14] On these premises it is conceivable that the orbicularis might be stimulated through associated control in a case of recent facial paralysis. This would probably not long be true. The oblique fibers of the orbicularis, acting on the tarsal curvature, may cause the extreme downward movement of the upper lid. This is difficult to visualize in a normal lid, and would be impossible with a paralyzed orbicularis. Under normal conditions the open eye is associated with tonic contraction of the levator palpebrae and inhibition of the orbicularis oculi. This relationship is reversed in sleep.

Following pathologic precedent, it is fair to presume that there is contracture of the orbicularis oculi when the facial nerve is paralyzed. Such contracture would affect the oblique fibers and would draw the upper lid downward in the event of relaxation of the levator palpebrae under the stimulation of the will to turn the eye downward. There is, however, no contracture in a case of recent facial palsy, although the downward movement of the lid is present.

In reporting the following cases I realize that the time which has elapsed since operation is too short to permit one to draw definite conclusions regarding return of function.

Case 1.—A radical mastoid operation was performed on the right ear of a patient on Aug. 18, 1932; this was followed by a second operation on the right ear on November 14. Complete facial paralysis appeared twelve hours after the operation. There had been a slight twitching at the time of operation. On

14. Beevor, C. E.: The Croonian lectures on muscular movements and their representation in the central nervous system, London, Adlard & Son, Ltd., 1904.

December 19, practically one month after the occurrence of the paralysis, the wound was reopened and the facial nerve was uncovered from the stylomastoid foramen to a point well forward of the knee. The middle third of the vertical portion of the nerve was found to be swollen and damaged. The nerve was freed from bony pressure well above and below the injured portion. It was found to be in continuity. The sheath was opened, and the nerve was covered by a Thiersch graft that extended forward to line the middle ear. The wound was immediately closed posteriorly except for a small tissue drain in the lower angle. The wound healed without incident, but troublesome granulation tissue in the middle ear still furnishes some secretion.

The facial paralysis was complete and unchanged when the patient was last seen, on May 29, 1933, more than five months after decompression.

Subsequent Note.—On Sept. 11, 1933, there was definite evidence of returning function of the paralyzed facial nerve.

Case 2.—A simple mastoid operation was performed on a patient on Feb. 24, 1933. A slight twitching occurred during the operation. Complete facial paralysis appeared nine hours after the operation. On March 8, decompression of the right facial nerve was performed. The electric bur was of assistance because of the unusual depth of the wound. There was considerable hemorrhage from the stylomastoid artery, which was controlled at the source. The nerve swelled on being released from the canal. It showed injury 2 mm. below the knee and for a distance of 4 mm. It was laid bare 4 mm. proximal to the wound and distal to the styloid mastoid foramen. The nerve was in continuity. The sheath was slit, and the posterior part of the wound was closed but for a small rubber drain in the lower angle. There was no return of function when the patient was last seen, on June 2, nearly three months after decompression.

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EFFECTS OF DRUGS ON VESTIBULAR REACTIONS

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The demand for some drug which will act specifically to reduce the irritability of the vestibular apparatus is great. For a long time general sedatives, such as bromides and opium derivatives, have been employed with some degree of success. The chief objection to these is that when a sufficient amount is prescribed to reduce vertigo, the patient is incapacitated by the general depression. Several years ago Jonkhoff [1] and de Kleyn and Versteegh [2] reported that the vestibular apparatus was decidedly influenced by a number of drugs. This led us [3] to study the effects of drugs on vestibular reactions. The outstanding compounds of each pharmacologic group were used. The nystagmic reaction to rotation was taken as the measure of the degree of stimulation of the vestibular system. As no operations were performed, there was no question of allowances for mutilation. The results were disappointing. There was but one drug that had any significant effect. Strychnine reduced the reaction. This was considered as due to a stimulation of the spinal cord.

Equilibrium is much more easily disturbed after one labyrinth is destroyed. In this condition there still remains the complete and healthy organ of equilibrium on one side. Therefore, it was thought that an animal with one labyrinth might show the effect of drugs when a normal animal would not.

The internal ears of dogs were destroyed by the operation described in one of our previous articles.[4] One labyrinth was left normal in each animal. After about a week, when equilibrium had been completely restored and the wound had healed, the tests were begun.

The animals were securely tied on a specially constructed board, so that the belly, throat and chin were in the same plane. This board was

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1. Jonkhoff, D. J.: Acta oto-laryng. **4**:174, 265 and 450, 1922.
2. de Kleyn, A., and Versteegh, C.: Arch. f. d. ges. Physiol. **196**:331, 1922.
3. Ross, E. L., and Fish, M. W.: Ann. Otol., Rhin. & Laryng. **38**:175, 1929. Ross, E. L.; Fish, M. W., and Olsen, A.: Effect of Some Drugs on the Vestibular Response to Rotation, Arch. Otolaryng. **14**:755 (Dec.) 1931.
4. Ross, E. L., and Olsen, A.: Effect of Destruction of One Labyrinth on Reactions to Rotation, Arch. Otolaryng. **17**:684 (May) 1933.

so constructed that it could be attached to a rotating machine in such a way that the axis of the body and head could be either horizontal or vertical. The rate of rotation and manner of sudden stop were the same as in our previous work. The duration of the nystagmus was measured with a stopwatch. Rotation was made in two ways. The spine and chin of the animal were always in a vertical position, and the spine was the approximate axis of rotation. When rotation was made to the right, the sagittal plane of the skull was tipped toward the shoulder approximately 45 degrees. This rotation had a maximum influence on the right

TABLE 1.—*Pentobarbital Sodium: Effect on Rotation Tests Performed on Dogs With One Labyrinth*

			Duration of Nystagmus in Seconds			
			Before Drug Vertical Rotation		After Drug Vertical Rotation	
Dog	Date	Labyrinth Destroyed	Right	Left	Right	Left
19	5/27/32	Right	10.6	14.2	5.6	7.8
		Right	8.4	14.2	....	10.4
		Right	9.8	15.2	5.2	12.8
Average		Right	9.6	14.5	5.4	10.3
20	5/30/32	Right	7.4	15.8	0.0	0.0
		Right	6.8	16.8	0.0	0.0
		Right	8.3	19.2	0.0	0.0
Average		Right	7.5	17.3	0.0	0.0
21	5/23/32	Right	12.0	15.3	12.4	7.6
		Right	11.7	15.8	10.2	
		Right	12.4	16.2	9.9	9.6
Average		Right	12.0	15.8	10.8	8.6
22	5/29/32	Right	8.0	13.8	0.0	0.0
		Right	9.2	17.5	0.0	0.0
		Right	8.4	13.2	0.0	0.0
Average		Right	8.5	14.8	0.0	0.0

anterior and left posterior vertical canals. When the rotation was to the left, the sagittal plane of the skull was tipped approximately 45 degrees toward the left shoulder. This rotation had a maximum action on the left anterior and right posterior vertical canals.

Tests were made on the animals every five minutes. After rotations had been made three times in each direction, the drug was injected intraperitoneally. Rotations at five minute intervals were continued till three tests were made in each direction.

The drugs used were those acting on the cerebrum, sympathetic nervous system and spinal cord. Pentobarbital sodium was used to depress the cerebrum and caffeine to stimulate it. Cocaine and epinephrine were employed to stimulate the endings of the sympathetic nerves. The spinal cord was stimulated with strychnine. The results with the different drugs are given in tables 1 to 8, inclusive. The average results with the various drugs are given in table 9.

TABLE 2.—*Pentobarbital Sodium: Effect on Rotation Tests Performed on Dogs With One Labyrinth*

Dog	Date	Labyrinth Destroyed	Duration of Nystagmus in Seconds: Before Drug Vertical Rotation, Right	Before Drug Vertical Rotation, Left	After Drug Vertical Rotation, Right	After Drug Vertical Rotation, Left
4	2/23/32	Left	19.9	10.2	11.9	Asleep
		Left	22.0	8.9	Asleep	Asleep
		Left	22.9	10.1	Asleep	Asleep
Average		Left	21.6	9.7	4.0	
7	2/12/32	Left	21.6	16.6	15.2	0.0
		Left	18.2	11.4	0.0	0.0
		Left	17.6	15.2	0.0	0.0
Average		Left	19.1	14.4	5.1	0.0
8	2/27/32	Left	13.6	7.7	11.0	0.0
		Left	14.3	8.0	0.0	0.0
		Left	14.0	9.6	0.0	0.0
Average		Left	14.0	8.4	3.6	0.0
9	3/ 5/32	Left	14.2	7.9	0.0	0.0
		Left	17.3	6.8	0.0	0.0
		Left	14.8	9.3	0.0	0.0
Average		Left	15.4	8.0	0.0	0.0
10	3/ 5/32	Left	26.2	10.3	21.1	8.9
		Left	24.6	8.3	20.0	8.0
		Left	24.8	9.4	20.4	9.2
Average		Left	25.2	9.3	20.5	8.7

TABLE 3.—*Caffeine: Effect on Rotation Tests Performed on Dogs with One Labyrinth*

Dog	Date	Labyrinth Destroyed	Duration of Nystagmus in Seconds: Before Drug Vertical Rotation, Right	Before Drug Vertical Rotation, Left	After Drug Vertical Rotation, Right	After Drug Vertical Rotation, Left
19	4/30/32	Right	8.1	16.1	....	14.8
		Right	6.4	15.4	....	14.8
		Right	5.6	16.2	....	15.2
Average		Right	6.7	15.9	....	14.9
20	5/ 9/32	Right	7.3	16.8	7.6	17.6
		Right	6.2	15.6	6.3	15.6
		Right	6.0	18.4	6.3	17.3
Average		Right	6.5	16.9	6.7	16.8
21	5/ 4/32	Right	10.2	16.8	10.6	15.5
		Right	10.4	17.5	11.4	15.0
		Right	10.2	17.2	....	....
Average		Right	10.3	17.2	11.0	15.2
22	5/13/32	Right	8.5	17.0	7.2	12.0
		Right	8.4	16.0	7.0	14.3
		Right	7.5	19.3	7.6	16.3
Average		Right	8.1	17.4	7.3	14.2
23	5/12/32	Right	9.3	14.8	9.4	13.5
		Right	7.8	15.6	8.8	12.8
		Right	9.9	16.5	8.2	14.2
Average		Right	9.0	15.6	8.8	13.5

TABLE 4.—*Caffeine: Effect on Rotation Tests Performed on Dogs with One Labyrinth*

Dog	Date	Labyrinth Destroyed	Duration of Nystagmus in Seconds: Before Drug Vertical Rotation, Right	Before Drug Vertical Rotation, Left	After Drug Vertical Rotation, Right	After Drug Vertical Rotation, Left
4	1/31/32	Left	19.5	12.2	17.6	8.8
		Left	17.7	11.2	18.2	10.1
		Left	23.2	10.6	20.3	10.8
Average		Left	20.1	11.3	18.7	9.9
8	2/29/32	Left	12.2	7.8	13.2	8.4
		Left	13.1	7.3	12.9	8.0
		Left	14.4	9.6	16.6	7.6
Average		Left	13.2	8.2	14.2	8.0
9	3/ 9/32	Left	13.6	7.6	13.8	8.0
		Left	12.9	....	17.0	11.0
		Left	17.2	7.2	14.3	9.6
Average		Left	14.6	7.4	15.0	9.5
10	3/ 9/32	Left	16.3	8.1	15.1	....
		Left	16.3	10.3	14.6	9.8
		Left	15.8	10.2	14.1	8.6
Average		Left	16.1	9.5	14.6	9.2

TABLE 5.—*Cocaine and Epinephrine: Effect on Rotation Tests Performed on Dogs with One Labyrinth*

Dog	Date	Labyrinth Destroyed	Duration of Nystagmus in Seconds: Before Drug Vertical Rotation, Right	Before Drug Vertical Rotation, Left	After Drug Vertical Rotation, Right	After Drug Vertical Rotation, Left
19	5/25/32	Right	12.4	13.0	10.5	16.2
		Right	11.0	15.8	9.2	16.9
		Right	8.8	15.7	9.0	15.6
Average		Right	10.7	14.8	9.6	16.2
20	5/29/32	Right	8.2	15.8	9.2	20.6
		Right	7.8	16.8	8.6	16.8
		Right	5.8	17.9	8.4	17.8
Average		Right	7.3	16.8	8.7	18.4
21	5/21/32	Right	10.4	14.6	11.2	14.7
		Right	13.8	17.2	12.7	14.2
		Right	13.0	14.5	12.5	14.9
Average		Right	12.4	15.4	12.1	14.6
22	5/24/32	Right	9.2	15.8	10.0	13.6
		Right	8.3	14.3	8.8	18.6
		Right	8.6	16.6	7.8	15.2
Average		Right	8.7	15.6	8.9	15.8

TABLE 6.—*Cocaine and Epinephrine: Effect on Rotation Tests Performed on Dogs with One Labyrinth*

Dog	Date	Labyrinth Destroyed	Duration of Nystagmus in Seconds: Before Drug Vertical Rotation, Right	Before Drug Vertical Rotation, Left	After Drug Vertical Rotation, Right	After Drug Vertical Rotation, Left
4	1/25/32	Left	19.1	15.2	21.4	11.2
		Left	18.0	11.8	21.2	12.6
		Left	15.8	13.8	23.8	8.5
Average		Left	17.6	13.6	22.1	10.8
7	2/13/32	Left	15.3	7.1	14.7	5.5
		Left	15.9	8.8	16.1	5.8
		Left	16.0	7.7	16.8	6.6
Average		Left	15.7	7.9	15.9	6.0
8	2/28/32	Left	13.0	7.1	11.6	7.0
		Left	15.2	8.4	13.2	8.4
		Left	14.8	7.5	12.6	7.2
Average		Left	14.3	7.7	12.5	7.5
9	3/ 7/32	Left	18.1	6.0	18.3	6.9
		Left	15.3	6.8	14.1	8.2
		Left	14.4	7.6	14.8	....
Average		Left	15.9	6.8	15.7	7.5
10	3/ 7/32	Left	21.0	8.0	17.5	7.4
		Left	19.0	8.4	17.8	7.2
		Left	18.8	7.6	18.6	7.2
Average		Left	19.6	8.0	18.0	7.3

TABLE 7.—*Strychnine: Effect on Rotation Tests Performed on Dogs with One Labyrinth*

Dog	Date	Labyrinth Destroyed	Duration of Nystagmus in Seconds: Before Drug Vertical Rotation, Right	Before Drug Vertical Rotation, Left	After Drug Vertical Rotation, Right	After Drug Vertical Rotation, Left
19	4/13/32	Right	15.5	25.6	8.0	19.6
		Right	12.1	20.4	8.7	17.6
		Right	13.8	16.8	8.4	19.3
Average		Right	13.8	20.9	8.4	18.8
20	4/11/32	Right	11.8	31.4	14.3	24.5
		Right	12.9	33.8	20.2	19.3
		Right	12.1	29.4	15.2	20.4
Average		Right	11.9	31.5	16.6	21.4
22	4/24/32	Right	7.3	26.4	8.1	18.4
		Right	6.9	18.6	6.9	17.8
		Right	6.7	21.8	6.3	18.5
Average		Right	7.0	22.3	7.1	18.2
23	4/25/32	Right	0.0	19.6	5.0	18.0
		Right	0.0	18.4	0.0	20.1
		Right	4.0	19.8	0.0	17.0
Average		Right	1.0	19.3	1.6	18.4

The reactions with pentobarbital sodium were variable. The drug often did away with the nystagmus entirely. The dogs in some cases were somewhat stuporous and reacted only slightly to any stimulus. From this we can conclude only that pentobarbital sodium reduced the vestibular response. There was no evidence that the action was specific.

TABLE 8.—*Strychnine: Effect on Rotation Tests Performed on Dogs with One Labyrinth*

Dog	Date	Labyrinth Destroyed	Duration of Nystagmus in Seconds: Before Drug Vertical Rotation, Right	Before Drug Vertical Rotation, Left	After Drug Vertical Rotation, Right	After Drug Vertical Rotation, Left
7	2/11/32	Left	19.8	10.8	24.7	7.0
		Left	18.1	11.8	17.6	6.6
		Left	20.1	11.2	16.5	7.6
Average		Left	19.3	11.3	19.6	7.1
8	2/25/32	Left	14.4	0.0	14.6	5.7
		Left	21.3	0.0	13.4	6.2
		Left	19.0	0.0	14.0	5.5
Average		Left	18.2	0.0	14.0	5.8
9	2/ 4/32	Left	19.8	12.1	16.4	0.0
		Left	21.2	7.8	16.2	5.6
		Left	19.0	6.4	16.9	6.3
Average		Left	20.0	8.8	16.5	4.0
10	3/ 4/32	Left	39.9	12.0	24.6	9.2
		Left	30.4	14.7	24.5	9.0
		Left	31.8	11.5	25.2	9.3
Average		Left	34.0	12.7	24.8	9.2

TABLE 9.—*Effect of Various Drugs on Rotation Tests Performed on Dogs With One Labyrinth: Averages Obtained from Tables 1 to 8*

Drug	Dogs	Labyrinth Destroyed	Duration of Nystagmus in Seconds: Before Drug, Rotation to Right	Before Drug, Rotation to Left	After Drug, Rotation to Right	After Drug, Rotation to Left
Pentobarbital sodium	4	Right	9.40	15.60	4.05	4.72
	5	Left	19.06	9.96	6.64	1.74
Caffeine	5	Right	8.12	16.60	6.76	14.92
	4	Left	16.00	9.10	15.62	9.15
Cocaine and epinephrine	4	Right	9.77	15.65	9.82	16.25
	5	Left	16.62	8.80	16.84	7.82
Strychnine	4	Right	8.42	23.50	8.52	19.20
	4	Left	22.87	10.93	18.72	6.52

Caffeine is a cerebral stimulant. Its action is mild compared with that of the opposite type of pentobarbital sodium. In only four of seventeen average tests were there increases in the reaction. In general, there was a small reduction in the duration of nystagmus resulting from rotation which was due to the action of caffeine. This agrees with our previous work done with caffeine on dogs with both labyrinths intact.

Stimulation of the endings of the sympathetic nerves with cocaine and epinephrine produced no consistent changes in the irritability of the single remaining labyrinth in this series of animals. This was true of both the positive and the negative stimulations. "Positive" and "negative" are terms applied to stimulations when the tendency of the endolymph is to move toward or away from the ampulla of a particular canal.

Strychnine, stimulating the spinal cord, decreased the reactions obtained by both positive and negative stimulation. In twelve of sixteen average tests there was a decrease. Of the eight cases in which the reaction was due to positive stimulation, only half showed a decrease. Of the eight tests made of the effectiveness of the drug on negative stimulation, there was a decrease in all but one. The average of all positive stimulations before administration of the drug was 9.68 seconds; after administration of the drug it was 7.52 seconds. The average of all the negative stimulations before strychnine was given was 23.18 seconds; after the drug had been given, the average was 18.96 seconds. In general, these animals showed a decrease of approximately 20 per cent in the reaction due to the influence of strychnine. This agrees with our previous work on dogs that had not been operated on.

SUMMARY AND CONCLUSIONS

A series of animals with but one labyrinth was given drugs representing the common pharmacologic groups. Pentobarbital sodium represented the cerebral depressants; caffeine, cerebral stimulants; cocaine and epinephrine, stimulants of the sympathetic nervous system, and strychnine stimulants of the cord. The change in the duration of the nystagmus after a standard rotation and quick stop was taken as the measure of the change in the irritability of the vestibular system.

The cerebral depressants decreased the reaction. However, the estimated degree of general depression was as great as, or greater than, the decrease of vestibular reaction. Caffeine did not increase the reaction to any significant degree.

Stimulation of the sympathetic nervous system by cocaine and epinephrine had no significant effect.

Strychnine, stimulating the spinal cord, decreased the vestibular reaction approximately 20 per cent.

SHOULD FUSOSPIROCHETAL INFECTIONS BE TREATED WITH ARSENICALS?

REPORT OF CASES

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In a recent article in *The Journal of the American Medical Association,* Lichtenberg, Werner and Lueck [1] questioned both the pathogenicity of the fusospirochetal organisms and the success of treatment with the arsenicals. After a number of years of experience with the disease in the laboratory and in the clinic, I have arrived at the conclusion diametrically opposed to that of Lichtenberg and his associates, that this group of anaerobic organisms has under certain conditions a primary pathogenicity, and that arsenical therapy is usually the most efficient treatment. In view of this difference of opinion, it seems advisable to review the evidence in favor of the pathogenicity of this group of organisms and to report some cases which illustrate the indications, dangers and limitations of arsenical therapy in fusospirochetal infections.

In 1894, Veillon [2] observed in necrotic, foul lesions of the throat the following mixture of organisms: cocci, Leptotriches vibriones, Spirochaeta denticola and fusiform bacilli. In December of the same year, Plaut [3] described Spirochaeta dentium and Miller's bacillus in pseudodiphtheritic angina. Following Vincent's [4] observations in 1896 and 1899, this type of infection was accepted as a clinical entity. The gradual development of knowledge of the disease has been reviewed in a recent monograph.[5]

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1. Lichtenberg, H. H.; Werner, M., and Lueck, E. V.: The Pathogenicity of the Fusiform Bacillus and Spirillum of Plaut-Vincent, J. A. M. A. **100**:707 (March 11) 1933.

2. Veillon, A.: Recherches sur l'étiologie et la pathogénie des angines aigües non diphtéritiques, Arch. de méd. expér. et d'anat. path. **6**:161, 1894.

3. Plaut, H. C.: Studien zur bacteriellen Diagnostik der Diphtherie und der Anginen, Deutsche med. Wchnschr. **20**:920 (Dec. 6) 1894.

4. Vincent, H.: Sur l'étiologie et sur les lesions anatomo-pathologiques, Ann. Inst. Pasteur **10**:488 (Sept.) 1896; Recherces bactériologiques sur l'angine á bacillus fusiformis, ibid. **13**:609 (Aug.) 1899.

5. Smith, D. T.: Oral Spirochetes and Related Organisms in Fuso-Spirochetal Disease, Baltimore, Williams & Wilkins Company, 1932.

In 1910, Ehrlich [6] was the first to report the use of an intravenously administered arsenic preparation in the treatment of fusospirochetal angina. Between 1911 and 1913, Sourdel,[7] Lebenthal,[8] Roger [9] and Rolleston [10] introduced the use of a glycerin solution of arsphenamine for local application in Vincent's angina (trench mouth), and this became the standard treatment in the World War (1914-1918).

EVIDENCE OF THE PRIMARY PATHOGENICITY OF FUSOSPIROCHETAL ORGANISMS

Clinical.—Spirochetes, fusiform bacilli, vibrios and cocci are present normally in practically every healthy mouth, beneath the tartar which collects in the gingival trough, where they are partially protected from the destructive effect of the free oxygen in the air. When the gums are inflamed by chemical or mechanical injury, spirochetes increase enormously in numbers, but they disappear completely when all the teeth are removed. The fact that these anaerobic organisms are normally present in the mouth cannot be used as a valid argument against their pathogenicity, since there are many examples of pathogenic organisms that live as saprophytes in the mouth and nasopharynx, e. g., pneumococci, meningococci, diphtheria bacilli and hemolytic streptococci. It is true that most oral infections in which fusospirochetal organisms are present clear up rapidly with simple treatment or without any treatment, as emphasized by Lichtenberg, Werner and Lueck,[1] but so do measles, pertussis and follicular tonsillitis in most cases and even in many cases, diphtheria. Lichtenberg and his co-workers produced spontaneous fusospirochetal infections in guinea-pigs by traumatizing the gums with a sterile scalpel and observed the development of infection in the throats of children in whom the trauma was incidental to removal of the tonsils. They seemed surprised that the infections healed readily; yet it is well known that most cases of staphylococcic and streptococcic cellulitis following trauma are also self-limited. It is not true, however, that all local fusospirochetal infections in the mouth heal satisfactorily.

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6. Ehrlich: Weitere Erfahrungen mit Ehrlich-Hata, München. med. Wchnschr. **57**:2268, 1910.

7. Sourdel, M.: Un cas d'angine de Vincent traité par la médication d'Ehrlich, Bull. gén. de thérap. **162**:818, 1911.

8. Lebenthal, D.: Essai de traitement local par l'arseno-benzol, Thèse de Paris, 1912, no. 273.

9. Roger, J. A. M.: Contribution à l'étude de l'action de l'arséno-benzol dans certaines affections spirillaires, Thèse de Paris, 1912, no. 257.

10. Rolleston, J. D.: Local Treatment of Vincent's Angina with Salvarsán, Proc. Roy. Soc. Med. **7**:1 (Oct.) 1914.

Zinserling [11] saw Vincent's infection of the pharynx extend into the soft tissues of the neck, with the formation of fatal Ludwig's angina. Fatal hemorrhage subsequent to Vincent's angina was reported by Bouty,[12] Dickerson,[13] Delsaux [14] and Cadhan.[15] Cadhan's patient was a physician, and the angina with subsequent fatal hemorrhage followed tonsillectomy. Hollender [16] observed in three patients severe fusospirochetal infection in the nose following submucous resection, which resulted in perforation of the septum. Ordinary antiseptics were of no value, although the lesion healed readily after treatment with arsenic. The patients were in good health prior to operation and did not show evidence of syphilis.

Perhaps the most common and most dangerous complication of Vincent's angina is the extension of the infection into the lungs. Fatal cases of this type have been reported by Bruce,[17] Sladen,[18] Gomez,[19] Lewis and Barenburg [20] and Zinserling.[11] I have seen Vincent's angina extend into the lungs in two patients and into the bronchi in one. Fortunately, the complication was recognized on the second, third and fourth days, respectively, and prompt recovery followed intravenous arsenical therapy.

Experimental.—In 1912, Marzinowski [21] produced Vincent's angina in a monkey by first scarifying the tonsil and then rubbing in a piece of membrane obtained from the tonsil of a man who had the infection. Typical angina with adenitis and enlargement of the lymph nodes developed, and the characteristic fusospirochetal organisms were demon-

11. Zinserling, W. D.: Ueber die fuso-spirochätöse Gangrän und einige verwandte Prozesse vorzugsweise bei Kindern (pathologisch-anatomische Beobachtungen), Jena, Gustav Fischer, 1928.

12. Bouty, R. T. C.: Vincent's Angina Among Troops, Brit. M. J. **2**:685 (Nov. 24) 1917.

13. Dickerson, W. E.: Fusospirillosis of Oropharynx, West Virginia M. J. **24**:119 (March) 1928.

14. Delsaux, V.: Contribution à l'étude des complications de l'angine fusospirillaire, Presse oto-laryng. belge **12**:145, 1913.

15. Cadhan, F. T.: Vincent's Disease, Canad. M. A. J. **17**:556 (April) 1927.

16. Hollender, A. R.: Delayed Healing of Septal Resections Due to Vincent's Infection: A Report of Three Cases, Arch. Otolaryng. **9**:422 (April) 1929.

17. Bruce, H. W.: Case of Vincent's Angina in Which the Larynx and Trachea Were Involved, Lancet **2**:1021 (Oct. 2) 1907.

18. Sladen, F. J., in discussion on Barker, L. F., and Miller, S. R.: Perforating Ulcer of the Hard Palate Resembling Tertiary Syphilis, J. A. M. A. **71**:797 (Sept. 7) 1918.

19. Gomez, F. R.: Pleuresia purulenta y gangrena pulmonar, Arch. latino-am. de pediat. **16**:656 (Aug.) 1922.

20. Lewis, J. M., and Barenburg, L. H.: Pulmonary Gangrene Due to Spirochetes and Fusiform Bacilli, Am. J. Dis. Child. **37**:35 (Feb.) 1929.

21. Marzinowski, E. M.: Inoculation expérimentale de l'angine de Vincent au Singe (Macacus rhesus), Compt. rend. Soc. de biol. **73**:389 (Oct.) 1912.

strated in both the membrane and an excised node from the neck. Davis and Pilot [22] produced a fatal empyema in rabbits by inoculating material from fusospirochetal angina directly into the pleural cavity. Kline [23] produced abscesses in the groins of guinea-pigs by introducing pieces of tonsillar membrane containing fusospirochetal organisms into previously traumatized tissues, and Smith [24a] obtained abscesses in the groins of guinea-pigs with similar material without having previously lowered the resistance of the tissues by trauma. In Smith's experiments, the infection could be transferred from guinea-pig to guinea-pig in series. Experimental pulmonary abscess and gangrene have been produced by many workers with fusospirochetal material obtained from one or more of the following sources: Vincent's angina, infected gums, sputum and even the lung itself. With fresh material from these sources pulmonary abscess has been produced in mice by Smith; [24b] in guinea-pigs by Smith; [24b] in rabbits by Kline [23] and Smith; [24b, c] in dogs by Crowe and Scarff,[25] Allen,[26] Hedblom, Joannides and Rosenthal,[27] Harkavy,[28] Van Allen, Adams and Hrdina [29] and Herrmann and Cutler.[30] Such a uniform series of positive experiments in a variety of animals by different investigators leaves little doubt that the whole fusospirochetal mixture has primary pathogenicity. These experiments, however, do not prove which organism or combination of organisms is essential for the reproduction of the disease.

From patients with the fusospirochetal type of pulmonary abscess, I have isolated the following organisms in pure culture: Treponema microdentium, Treponema macrodentium, Treponema buccale, Vibrio viridans, Bacterium melaninogenicum, Micrococcus gazogenes, large

22. Davis, D. J., and Pilot, I.: Studies of Bacillus Fusiformis and Vincent's Spirochete, J. A. M. A. **79**:949 (Sept. 16) 1922.

23. Kline, B. S.: Experimental Gangrene, J. Infect. Dis. **32**:481 (June) 1923.

24. Smith, D. T.: (*a*) Fusospirochetal Disease of the Lungs Produced with Cultures from Vincent's Angina, J. Infect. Dis. **46**:303 (April) 1930; (*b*) Experimental Aspiratory Abscess, Arch. Surg. **14**:231 (Jan., pt. 2) 1927; (*c*) Etiology of Primary Bronchiectasis, ibid. **21**:1173 (Dec.) 1930.

25. Crowe, S. J., and Scarff, J. E.: Experimental Production of Lung Abscess in the Dog, Internat. S. Digest **3**:323 (June) 1927.

26. Allen, D. S.: Etiology of Abscess of the Lung, Arch. Surg. **16**:179 (Jan., pt. 2) 1928.

27. Hedblom, C. A.; Joannides, M., and Rosenthal, S.: Pulmonary Abscess: Experimental Study, Ann. Surg. **88**:823 (Nov.) 1928.

28. Harkavy, J.: Pathogenesis of Aspiratory Abscess of Lung: Its Possible Relation to Abscess of Lung Following Tonsillectomy, Arch. Int. Med. **43**:767 (June) 1929.

29. Van Allen, C. M.; Adams, W. E., and Hrdina, L. S.: Bronchogenic Contamination in Embolic Abscess of the Lungs, Arch. Surg. **19**:1262 (Dec., pt. 2) 1929.

30. Herrmann, L. G., and Cutler, E. C.: The Experimental Production of Chronic Abscess of the Lung, J. Clin. Investigation **7**:501 (Aug.) 1929.

fusiform bacilli, small fusiform bacilli, alpha, beta and gamma streptococci and various types of staphylococci. The cultures of Treponema vincenti and Spirillum sputigenum were mixed with cocci. Some of the strains of streptococci produced fatal hemorrhagic cellulitis and pneumonitis, and some of the staphylococci caused small nonfetid abscesses. None of the other organisms initiated infection when inoculated into animals. Neither the spirochetes alone nor those combined with fusiform bacilli could infect mice, guinea-pigs or rabbits. However, a mixture made from pure cultures of T. microdentium, Vibrio viridans, a small fusiform bacillus and a hemolytic streptococcus produced a typically foul, necrotic abscess which could be transmitted from animal to animal in series.[24a] The importance of the spirochete in this symbiotic mixture was demonstrated by the observation that the infection could no longer be transferred if the spirochetes disappeared either spontaneously or as a result of the treatment with sulpharsphenamine, although all the other organisms were present. Herrmann and Cutler [30] likewise failed to produce chronic fetid abscesses in dogs with any one of twelve varieties of organisms isolated in pure culture from pulmonary abscesses, although a mixture of the anaerobic forms caused a chronic, foul abscess resembling the true chronic abscess in man.

ARSENICAL THERAPY IN FUSOSPIROCHETAL INFECTIONS

Treatment of Vincent's Angina in Patients Receiving Antisyphilitic Treatment.—In general, patients receiving antisyphilitic treatment have more inflammation of the gums and more fusospirochetal organisms in the mouth while receiving mercury, and improve in a striking manner when the treatment is changed to intravenous administration of arsenicals. At times, however, as reported by Sutton,[31] Williams,[32] and Hillsman and Driscoll,[33] acute Vincent's angina develops while the patient is receiving intravenous arsenicals. It is possible that the spirochetes have become arsenic-fast. Driscoll [34] obtained rapid recovery in such patients by stopping the arsenicals and giving intravenous antimony and potassium tartrate, which is used extensively in South America for the nonsyphilitic spirochetal infections.[35]

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31. Sutton, I. C.: Vincent's Angina Occurring in a Patient Under Treatment for Syphilis, J. A. M. A. **83**:1919 (Dec. 13) 1924.

32. Williams, G. A.: Vincent's Angina: A Case Occurring During Antisyphilitic Treatment, Arch. Dermat. & Syph. **20**:322 (Sept.) 1929.

33. Hillsman, J. A., and Driscoll, T. L.: Fuso-Spirillary Infection Which Developed During Active Arsphenamine, Virginia M. Monthly **52**:312 (Aug.) 1925.

34. Driscoll, T. L.: Treatment of Vincent's Angina, Virginia M. Monthly **51**:233 (July) 1924.

35. Risquez, J. R.: La espiroquetosis bronquial en Venezuela, Gaz. med. de Caracas **30**:193, 209, 225, 241, 257 and 273 (July) 1923.

The failure of Lichtenberg and his associates to influence with sulpharsphenamine the experimental oral fusospirochetal infections in guinea-pigs may have been due to the lowering of the resistance of the animals by the large doses of arsenic which they used. Kolmer [36] found that if excessive doses of arsenicals were employed in experimental syphilis in rabbits, the animals were definitely less resistant than those receiving smaller doses. Lichtenberg and his co-workers gave sulpharsphenamine, 25 mg. per kilogram of body weight, on alternate days for four doses, or a total of 100 mg. per kilogram in eight days. In some of my own experiments in which fusospirochetal abscesses were produced in the groins of guinea-pigs with a mixture made from pure cultures of T. microdentium, a small fusiform bacillus, an anaerobic hemolytic streptococcus and a vibrio, a single dose of 10 mg. of sulpharsphenamine per kilogram of body weight was sufficient to reduce materially the infection in the guinea-pigs and render the material incapable of producing an abscess when transferred to another guinea-pig. The untreated controls presented large, foul, necrotic abscesses, and the infection could be transmitted without difficulty to new animals.

Severe Vincent's Angina in Children.—Mild cases of Vincent's angina in children may clear up with local treatment or without treatment, as shown by Lichtenberg and his associates. The following case illustrates the failure of local treatment in the severe type of infection. The patient continued to grow worse in spite of local treatment over a period of five days, but responded to intramuscular injections of sulpharsphenamine.

CASE 1.—D. J. C., a girl, aged 2 years, acquired a sore mouth six days before admission. The family physician found ulcers on the tongue and in the pharynx; he made a diagnosis of Vincent's angina and prescribed local treatment. The oral lesions grew steadily worse, the temperature reached 38 C. (100.4 F.), and the child remained fretful and cross and refused food. When she was admitted to the hospital on Sept. 29, 1932, the gums were very red, and small white patches were seen over them, as well as over the mucous membrane of the cheeks and the inner surface of the lips. The tonsils and palate were covered with a large grayish membrane which contained large numbers of characteristic fusospirochetal organisms. Except for large, tender submaxillary and anterior cervical glands, the results of the general physical examination were negative.

A diet of soft foods was given which contained large amounts of orange juice. Sulpharsphenamine (0.2 Gm.) was administered intramuscularly on September 29 and on October 1, 2 and 3. There was marked improvement within twenty-four hours after the first injection, and the child was discharged four days after admission, well except for slight congestion of the pharynx. One or, at the most, two injections of sulpharsphenamine should have been sufficient to cure the patient.

Chronic Fusospirochetal Infections of the Larynx.—Chronic infections of the larynx with fusospirochetal organisms which do not respond to local applications should be treated with intravenous arsenicals.

36. Kolmer, J. A.: Principles and Practice of Chemotherapy, Philadelphia, W. B. Saunders Company, 1926.

Case 2.—Four months before admission to the hospital, R. W., a white man, aged 35, experienced a raw feeling in the throat and pain on speaking or swallowing. He found that whispering reduced the amount of pain and he therefore adopted that mode of speech. When he was admitted to the hospital on Jan. 14, 1932, the tongue, mouth and pharynx appeared to be normal, but examination of the larynx revealed a membrane over the false cords and generalized redness and edema. Smears from the larynx contained typical fusospirochetal organisms. The findings in the remainder of the physical examination were essentially normal.

While in the hospital, the patient received the regular diet given in the ward and 0.3 Gm. of neoarphenamine intravenously on January 16 and 18; 0.6 Gm. on January 22, and 0.45 Gm. on January 26. One week after admission the pain disappeared, and the patient began to use his voice. Laryngoscopic examination at the time of discharge demonstrated that the larynx was normal.

Chronic Trench Mouth.—Patients with acute or subacute trench mouth who do not improve with local treatment should receive intravenous arsenicals. Although this type of therapy has been employed for years by many dentists and physicians in the treatment of trench mouth and pyorrhea, it has not been adopted by the more conservative members of the two professions because the ultimate results were not satisfactory even when the immediate response was very dramatic. My results were not good until I began to increase the supply of calcium and vitamins in the diet and insisted on thorough dental treatment while the patient was receiving treatment with arsenic. Frequently, in the milder cases, local dental treatment, supplemented by a diet rich in calcium and vitamins, cures the patient.

Case 3.—M. N. K., a white woman, aged 39, was referred by Dr. L. M. Edwards, a dental surgeon, with the history of chronic suppurative gingivitis. In addition to the disease of the gums, the voice had been husky for the past three months. Examination of the mouth on Jan. 6, 1933, showed generalized redness of the gums, many old pockets of pyorrhea, and marked congestion and some edema of the larynx. Smears from the mouth showed typical fusospirochetal organisms. The results of the general physical examination and the other laboratory examinations were essentially negative.

The patient's diet was supplemented daily with three glasses of milk, two glasses of orange juice and 12 minims of halibut liver oil with viosterol 250D. She received sulpharsphenamine, 0.3 Gm. intramuscularly, on January 6, 13 and 20. The extreme redness of the gums disappeared one week after the first injection. Following the second dose of sulpharsphenamine the teeth were thoroughly cleaned, and the pockets of pyorrhea were treated locally by Dr. Edwards. After the third injection the larynx returned to normal, and the huskiness of the voice disappeared. The patient was discharged on January 27, with the mouth in a satisfactory condition except for the areas of destruction between the teeth.

Vincent's Infection in Avitaminosis.—Severe fusospirochetal infection of the mouth and the vagina occurring in patients with avitaminosis should be treated with intravenous arsenicals in addition to a diet rich in vitamins.

CASE 4.—I. D., a Negress, aged 21, was admitted to the hospital on Feb. 26, 1932, with a history of sore mouth for two weeks and infection of the vagina for one week. The patient appeared ill, with a temperature of 39 C. (102.2 F.); the pulse rate was 140, and the respiratory rate, 40. The tongue, buccal mucosa and pharynx were intensely red and covered with patches of membrane which on smear showed large numbers of typical fusospirochetal organisms. The cervical lymph nodes were enlarged and tender, and a moderate number of scattered râles were heard throughout the lungs. The patient had profuse leukorrhea, an inflamed vaginal mucosa with formation of membrane resembling that in the mouth and subacute bilateral salpingitis with a cystic left ovary. Smears made from the vagina twenty-four hours after the first injection of neoarsphenamine contained fusiform bacilli, vibrios and cocci, but no spirochetes. She had been eating the kind of diet which predisposes to pellagra, and although there were no cutaneous lesions, diarrhea or dementia, the presence of pellagra was probable but not proved.

She was given a liquid diet containing a large amount of milk, tomato juice and orange juice and fluids by hypodermoclysis. The mouth and vagina were irrigated with a 2 per cent solution of sodium perborate, and neoarsphenamine, 0.45 Gm., was administered on February 27 and March 1 and 12. There was gradual but steady improvement in the general condition of the patient as well as in the appearance of the oral and vaginal lesions. She was discharged on March 17, with a temperature of 37.4 C. (99.3 F.), a pulse rate of 100, and a respiratory rate of 24, to continue local treatment at home.

Postoperative Pulmonary Infections.—Postoperative pulmonary complications with fusospirochetal organisms should be treated promptly with intravenous arsenicals. Although the infection in many cases clears up without such treatment, in some fatal abscess or gangrene develops and hence the danger of doing harm with the arsenic seems less than that of allowing the infection to run its course.

CASE 5.—T. M., a white man, aged 20, was admitted to the hospital on Sept. 30, 1931, for repair of a bilateral inguinal hernia. The operation was performed with the patient under nitrous oxide and oxygen anesthesia on October 2. Twenty-four hours following operation his temperature was 38 C. (100.4 F.), and he became dyspneic. Forty-eight hours after operation there was marked cyanosis, and the temperature was 39 C. (102.2 F.), the pulse rate 120, and the respirations, 42. Atelectasis of the right middle and right lower lobes was demonstrated by physical examination and by roentgenogram. Hyperventilation with 5 per cent carbon dioxide and 95 per cent oxygen was induced on two occasions on October 4. Sputum obtained after the inhalation was small in amount but purulent, and it contained spirochetes, fusiform bacilli, vibrios and cocci. The lungs expanded rapidly after hyperventilation, but moist râles were still present over the right lower lobe posteriorly, and the roentgenogram showed a fine mottling in this area. Neoarsphenamine (0.45 Gm.) was given on October 5. The temperature, pulse and respirations, which had already begun to return to normal following the inhalation, returned rapidly to normal, and the râles disappeared from the lungs in forty-eight hours. The patient was discharged as well on October 20.

Pulmonary Abscess.—Patients with pulmonary abscess who have fusospirochetal organisms in the sputum should receive intravenous treatment with arsenicals. Of 940 such persons, reports of whose cases

were collected from the American literature, and to whom only general medical care was given, 382, or 40 per cent, died.[4] With arsenical therapy in addition to general medical care, Kline and Berger[37] reported recovery in 11 of 15 consecutive cases of acute fusospirochetal abscess and Smith[38] observed recovery in 13 of 14 patients whose illness was diagnosed and who were treated between the third day and the eighth week of the disease. Chronic abscesses of three or more months' duration are rarely if ever cured with arsenic, although many are improved and the patients made better operative risks.

CASE 6.—L. W., a Negro, aged 23, was admitted to the hospital on Feb. 18, 1931, complaining of cough, expectoration, night sweats, weakness and dyspnea growing gradually worse for the past five weeks. The patient was obviously ill, with a temperature of 40.4 C. (104.7 F.), a pulse rate of 130 and a respiratory rate of 36. The physical and roentgenographic examinations revealed a typical pulmonary abscess involving the middle half of the right lung. The sputum was green, purulent and foul, and showed on examination with the dark-field apparatus large numbers of spirochetes, fusiform bacilli, vibrios and cocci. Repeated examinations failed to reveal tubercle bacilli.

The patient was given a liquid diet, supplemented by cod liver oil and tomato juice, and neoarsphenamine, 0.3 Gm., on February 20, 24 and 28 and on March 5. There was rapid improvement in his condition, the temperature, pulse and respirations becoming normal on the fifth day after the first injection of arsenic. The sputum, which varied in amount from 200 to 300 cc. on admission, rapidly diminished to 50 cc. or less within five days and entirely disappeared after three weeks. The patient was discharged on April 7 in good condition. Reexamination and roentgenograms on May 5 showed that the lungs were normal.

COMMENT

Arsenicals administered intravenously are not harmless. It is well known that severe and at times fatal reactions follow their administration; hence they should never be used except when clearly indicated. As emphasized by Lichtenberg and his associates,[10] mild fusospirochetal infections frequently are self-limited, and many of the more severe infections respond to local treatment. However, intravenous treatment with arsenicals is indicated in patients who do not respond to local treatment and in those whose lungs have become involved. Small doses of from 0.2 to 0.3 Gm. repeated at intervals of from two to four days are less dangerous and quite as effective as the larger doses.

CONCLUSIONS

1. There is clinical evidence for the belief that the fusospirochetal mixture of anaerobic organisms is pathogenic.

37. Kline, B. S., and Berger, S. S.: Pulmonary Abscess and Pulmonary Gangrene, Arch. Surg. **18**:481 (Jan., pt. 2) 1929.

38. Smith, D. T.: Diagnosis and Medical Treatment of Incipient Pulmonary Abscess, South. Med. & Surg. **94**:149 (March) 1932.

2. If one can consider a group of anaerobic organisms acting in symbiosis as a single entity, Koch's postulates have been fulfilled: (*a*) The fusospirochetal organisms are constantly present in the lesion; (*b*) they have been isolated in pure culture; (*c*) when recombined and injected into animals, they produced the typical disease; (*d*) they have been isolated from the experimental lesions; (*e*) up to the present, the typical disease has not been produced by any other organism or any other combination of organisms.

3. The use of intravenous injections of neoarsphenamine or sulpharsphenamine is the most effective treatment for the more severe and the chronic infections with fusospirochetal organisms.

CHEMICAL COMPOSITION OF PUS FROM SINUSES

FURTHER OBSERVATIONS

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In 1931, in a series of forty-eight patients, we [1] investigated sinus disease from the standpoint of blood chemistry, composition of sinus bone and calcification of membranes and included two determinations of the calcium and phosphorus content of sinus pus. Since that time, further work has been done on the chemical composition of sinus pus, as it was thought that additional data might be obtained that would prove of value in the diagnosis and prognosis of sinus disease as well as in the determination of the progress or advancing chronicity.

It was thought, too, that if any further light on the subject could be revealed, to give a better understanding of sinus disease by the elimination of some of the confusion existing with reference to the therapeutic necessities to bring about relief, the time and effort put forth in this study would be amply justified.

The possibility of using the calcium concentration of pus as a means of diagnosis or prognosis in pathologic conditions involving necrosis of bone was suggseted a few years ago by Friesner and Rosen.[2] They employed this procedure for determining the proper time for surgical intervention in mastoiditis. Analyses of pus obtained from the aural canal in twenty-four cases of acute mastoiditis with necrosis of bone showed from 21 to 58 mg. of calcium per hundred cubic centimeters of pus. These values were considerably higher than those obtained from tissues that did not involve bony structure, such as abscesses of the liver, lungs and chest.

As a basis for the determination of an increased or diminished amount of calcium in pus, we utilized the averages found in a series of specimens of pus which we knew to be from parts wherein definite

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From the Rice Institute (H. O. Nicholas).

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1. Israel, S., and Nicholas, H. O.: The Biochemical Study of Sinus Disease, J. A. M. A. **97**:1453 (Nov. 14) 1931.

2. Friesner, I., and Rosen, S.: (*a*) Calcium Content of Pus, J. A. M. A. **88**:1231 (April 16) 1927; (*b*) A New Aid in the Diagnosis of Mastoiditis, Arch. Otolaryng. **7**:317 (April) 1928.

involvement of bone was present and then in specimens of pus obtained from abscesses of soft tissue in various parts of the body.

The average amounts of calcium and phosphorus obtained from these specimens with and without involvement of bone permit of a fair and impartial estimation in arriving at a conclusion with reference to an increased or diminished calcium and phosphorus content in pus with or without involvement of bone. For these determinations, the accepted tests available at the present time were utilized; they showed that in pus from abscesses of soft tissue the average calcium content is 16.5 per cent and the phosphorus content, 37.5 per cent; these values are typical of abscesses of soft parts without osseous involvement, and show a definitely higher ratio of phosphorus to calcium.

The patients represented in this study varied in age from 10 to 65 years and consisted of both males and females. Only patients with definite evidence of sinus disease in the history and in physical and roentgen examination were included. Some of the patients have been kept under observation over a long period.

The majority of the specimens of pus were collected from the nasal passages by means of a specially made pipet under aspiration, and the pus was immediately expressed into small (50 by 9 mm.) glass test tubes, which had been weighed previously. The tubes containing the pus were again weighed, and the pus was transferred to 10 cc. volumetric flasks by means of distilled water, five 1 cc. portions being used in the transfer. The flask was then filled to the mark with a 16 per cent solution of trichloracetic acid. The samples were allowed to stand, with frequent shaking, for at least forty-eight hours before the precipitated protein was filtered off. Aliquots of the filtrate representing between 50 and 150 mg. of pus were used for the determinations of calcium and phosphorus.

Calcium was determined according to the method of Roe and Kahn,[3] except that all reagents were reduced to one-fifth volume and the final volume of 3 cc. was read in a colorimeter using 1 cc. micro cups. When the amount of material permitted, duplicate analyses were made by ashing a 1 cc. sample and determining the calcium by means of the Kramer and Tisdall[4] potassium permanganate titration method. Good agreement between the results obtained by the two methods was observed in all cases. Inorganic phosphorus was determined by the method of Fiske and Subbarow,[5] one-fifth volume of the reagents again

3. Roe, J. H., and Kahn, B. S.: The Colorimetric Determination of Blood Calcium, J. Biol. Chem. **81**:1, 1929.

4. Kramer, B., and Tisdall, F. F.: A Simple Technique for the Determination of Calcium and Magnesium in Small Amounts of Serum, J. Biol. Chem. **47**:475 (Aug.) 1921.

5. Fiske, C. H., and Subbarow, Y.: The Colorimetric Determination of Phosphorus, J. Biol. Chem. **66**:375 (Dec.) 1925.

being used and the colorimetric readings being made in the 1 cc. colorimetric cups.

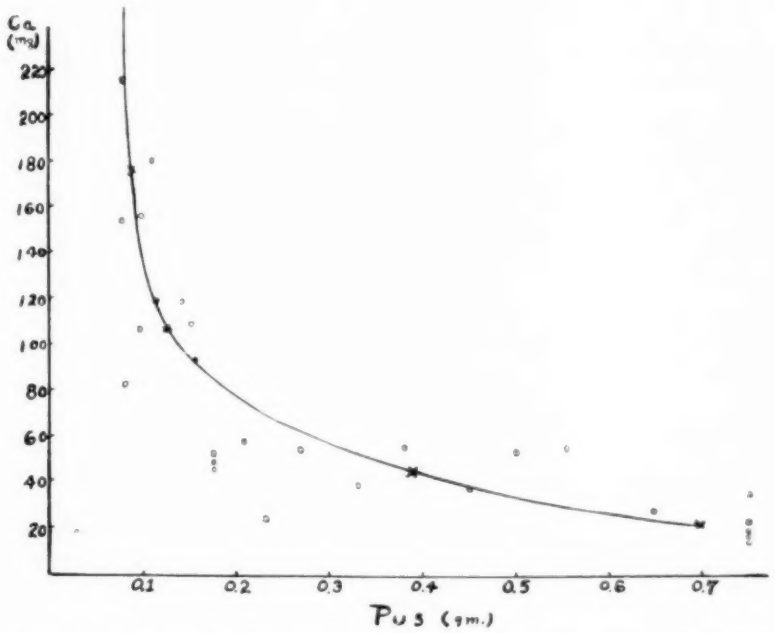
Table 1 gives the data on twenty-seven specimens from patients who exhibited frank sinus disease. The majority of specimens were obtained from the nose; a few were obtained directly from the sinus cavity at operation. It will be noted that the calcium content varied widely, from 14 to 215 mg. of calcium per hundred grams of pus, with an average value of 73 mg. In general, the calcium content (average, 73 mg.) was greater than the phosphorus content (average, 23 mg.), there being only four exceptions to this.

TABLE 1.—*Calcium and Phosphorus in Sinus Pus*

Case	Weight of Sample, Gm.	Calcium, Mg.	Phosphorus, Mg.	Source of Specimen
1	0.073	215	40	Nasal
2	0.090	155	29	Nasal
3	0.072	153	35	Nasal
4	0.110	118	45	Nasal
5	0.134	118	20	Nasal
6	0.156	109	16	Nasal
7	0.087	106	35	Nasal
8	0.156	93	29	Nasal
9	0.080	81	31	Nasal
10	0.214	58	8	Nasal
11	0.565	55	20	Nasal
12	0.387	55	13	Nasal
13	0.500	53	36	Nasal
14	0.176	51	8	Nasal
15	0.179	46	10	Nasal
16	0.178	45	11	Nasal
17	0.328	38	24	Nasal
18	0.645	27	7	Nasal
19	0.231	23	16	Nasal
20	1.544	21	8	Nasal
21	0.114	180	22	Oeprative
22	0.270	53	9	Operative
23	0.449	37	32	Operative
24	1.500	35	49	Operative
25	1.0	19	21	Operative
26	10.0	16	50	Operative
27	4.0	14	19	Operative

A noticeable observation that we are at a loss to explain at the present time is the relatively low calcium content in specimens collected direct from the sinuses at the time of operation as compared to the specimens aspirated from the nose. Theoretically, this might be explained by assuming that the pus which is collected as it drains through the nasal passages becomes concentrated, owing to evaporation, resulting in an increased concentration of calcium. However, if this were purely a process of evaporation, one would expect the phosphorus content to be changed similarly, which is not the rule, for most of the specimens of pus obtained at operation show a relatively higher content of phosphorus than of calcium.

If the weight of the specimen of pus is plotted against the calcium concentration, as in the chart, a rather uniform relationship (within certain limits) can be seen. These twenty-seven cases fall into four



Relationship of the calcium concentration to the amount of pus. The curve is drawn through the points marked *X*, which are the average values for the four groups, as shown in table 2.

TABLE 2.—*Relationship of Amount of Pus to Calcium Concentration*

Group	Number of Cases	Calcium, Mg.	Amount of Pus, Gm.	Calcium Concentration × Amount of Pus = Constant
1.....	4	153-215 (176)*	0.072-0.114 (0.087)	176 × 0.087 = 15
2.....	6	81-118 (104)	0.080-0.156 (0.120)	106 × 0.121 = 13
3.....	12	23-58 (45)	0.176-0.645 (0.387)	45 × 0.387 = 17
4.....	5	14-35 (21)	More than 0.7	21 × 0.71 † = 15

* The figures in parentheses represent the average values.
† The value 0.7 was calculated by assuming that 15 is the average value of the constant, as obtained in the other groups.

groups, as shown in table 2. There seems to be an inverse relationship between the amount of the specimen and the calcium concentration in those cases in which the sample is less than 0.7 Gm. (i. e., 0.7 cc.). However, in group 4, which includes four of the seven operative

specimens, this relationship is lost, possibly as the result of evaporation. The possibility of such a factor did not occur to us until recently and therefore needs further elucidation before a complete understanding can be had.

Table 2 shows that the greatest number of specimens fall in group 3, with a range of from 23 to 58 mg. of calcium, or an average of 45 mg. The calcium content in group 4, the lowest, was found to be from 14 to 35 mg., or an average of 21 mg.

Table 3 gives the results obtained on eight control specimens of nonsinus pus, the values corresponding favorably with those given by Friesner and Rosen.[2a] The calcium content is rather low and the phosphorus content rather high as compared to those of sinus pus. It is also interesting to note in table 3 that case 1, representing a specimen taken from severe osteomyelitis of the tibia, showed a high calcium content, as one would expect with involvement of bone. This is in contrast with

TABLE 3.—*Calcium and Phosphorus in Nonsinus Pus*

Case	Weight of Sample, Gm.	Calcium, Mg.	Phosphorus, Mg.	Source of Specimen
1	0.125	120	26	Osteomyelitis
2	0.500	16	20	Osteomyelitis
3	0.600	19	100	Rectal abscess
4	0.610	27	41	Abscess of liver
5	1.387	14	36	Periurethral abscess
6	2.438	15	12	Empyema
7	2.0	12	11	Empyema
8	5.0	12	25	Culdesac of Douglas

case 2, also a case of osteomyelitis, though a larger original amount by weight was examined, which showed a low calcium content and a high phosphorus: calcium ratio, findings which are more in keeping with pus from an abscess of soft tissue without involvement of bone.

Table 3 also shows, with the exception of cases 1, 2, 6 and 7, findings typical of abscesses of the soft parts, namely, a higher content of phosphorus than of calcium. Cases 6 and 7 represent specimens from empyema of the chest and show that the specimens examined were greater in quantity than those from some of the other abscesses of soft tissue and had a slightly higher calcium content than phosphorus content, both of which, however, were rather low. Whether or not there was any involvement of bone in these instances we are unable to state.

In the majority of instances, the patients with the most severe involvement and the most chronicity showed a high or noteworthy increase in the calcium content of the pus.

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MUCORMYCOSIS OF THE MAXILLARY SINUS

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A fungus was discovered in pus washed from the maxillary sinuses of a patient during the routine examination of such material. The forms of this organism first encountered resembled body cells so closely that their significance was passed over until forms encountered later made their identity certain. This organism was observed over a period of several months during which time it underwent certain morphologic changes. It was exposed to antiseptic solutions which were instilled into the sinuses in an effort to eradicate the infection. The presence of chronic inflammatory changes in the nasal membranes associated with this unusual type of infection is of considerable interest.

There is some evidence to the effect that fungi may cause pathologic changes in the membranes of the nasal fossa. Duverger [1] reported a case of aspergillosis of the nasal fossa associated with changes resembling caseous coryza (ozena). Dunn [2] and later Deile [3] described somewhat similar cases of Aspergillus fumigatus infection. Zarniko [4] reported a case of a woman, aged 50, with occlusion of the nose due to aspergillosis. Siebenman [5] described an infection of the nasal fossae with Aspergillus nidulans and fumigatus, together with Mucor corymbifer. Rhinosporidiosis is defined in Manson's [6] "Tropical Diseases" as, "A disease due to a yeast like organism, Rhinosporidium Seeberi, which infects the mucous membrane of the nose, producing nasal polypi and tumors of the cheek, the conjunctiva, lacrymal sac, and uvula." In a recent study of fungi of the nasal cavities, Motta [7] found that fungi were present in 16.5 per cent of 36 normal noses studied. Organisms of different varieties were recovered, and of these only Cryptococcus

1. Duverger, J.: Aspergillose des fosses nasales et coryza caséeux, Rev. hebd. de laryng. **1**:641, 1906.

2. Dunn, J.: Growth of the Aspergillus Glaucus in the Human Nose, Ztschr. f. Ohrenh. **29**:95, 1896.

3. Deile, R.: Ansiedelung von Aspergillus fumigatus in beiden Nasenhöhlen bei Ozäna, Ztschr. f. Ohrenh. **46**:386, 1904.

4. Zarniko, C.: Aspergillusmykose der Kieferhohle, Zahnärztl. Wchnbl., Hamb. **5**:149 and 155, 1891.

5. Siebenman, cited by Duverger.[1]

6. Manson-Bahr, P. H.: Manson's Tropical Diseases, New York, William Wood & Company, 1931.

7. Motta, R.: Fungi of Nasal Cavity in Abnormal and Pathologic Conditions, Bull. e. atti d. r. Accad. med. di Roma **4**:240, 1929.

was encountered twice. He showed that fungi of the open air are the ones generally found in the nose, and also showed that fungi were more often found in noses with definite pathologic conditions present. Castellani and Chalmers,[8] under the title of "Mucormycosis of the Nose," stated: "Special cases are on record in which species of Mucoraceae (generally Lichtheimia Corymbifera) were found in various affections of the nose. Their pathogenic rôle in such affections has not been demonstrated with certainty."

In a recent study of actinomycosis of the head and neck in relation to intracranial and cranial complications, MacFee [9] reported infections of these regions, but he did not mention primary infection of the sinuses. Prior to this, New [10] collected the report of 107 cases of actinomycosis of the head and neck. He did not mention primary infection of the sinuses, but stated that cases of actinomycosis occur in which there is involvement of almost the entire half of the head and neck. Iglauer [11] reported two cases of pneumocele of the frontal sinus, probably secondary to blastomycosis.

Other fungi so far reported as being the causative agent in infections of the accessory nasal sinuses have been in the family of Aspergillus. Tilley [12] reported 5 cases of infection of the maxillary sinus with this organism. It is of interest that in his first case the condition was diagnosed from the appearance of the tissues in microscopic section as endothelioma myxomatodes. He found that the radical Caldwell-Luc operation relieved the symptom. In Harmer's [13] case, the symptoms of hay fever were prominent. Membranous material was washed from the maxillary sinus which on culture yielded Aspergillus fumigatus. Intranasal drainage of the sinus effected relief from symptoms. Skillern [14] washed from the maxillary sinus material which resembled cottage cheese. Aspergillus, probably of the species fumigatus, was identi-

8. Castellani, A., and Chalmers, A. J.: Manual of Tropical Medicine, ed. 3, London, Baillière, Tindall & Cox, 1919, p. 976.

9. MacFee, W. F.: Actinomycosis of Head and Neck with Intracranial and Pulmonary Metastases, S. Clin. North America **12**:427 (April) 1932.

10. New, G. B.: Actinomycosis of Head and Neck, Tr. Am. Acad. Ophth. **28**:392, 1923.

11. Iglauer, S.: Pneumocele of Frontal Sinus with Report of Case, Probably Secondary to Blastomycosis, Ann. Otol., Rhin. & Laryng. **29**: 56, 1930; Tr. Am. Laryng., Rhin. & Otol. Soc. **36**:414, 1930.

12. Tilley, H.: Aspergillosis of Maxillary Antrum, Proc. Roy. Soc. Med. (Sect. Laryng.) **8**:14, 1914-1915.

13. Harmer, D., and Jockes, T.: Specimens of Aspergillus Fumigatus from Nasal Sinuses, Proc. Roy. Soc. Med. (Sect. Laryng.) **12**:187, 1918-1919.

14. Skillern, R. H.: Aspergillosis of the Maxillary Sinus, Laryngoscope **31**: 946, 1921.

fied in this material. Mackenzie [15] reported a case of infection of the maxillary sinus by Aspergillus fumigatus. Recently, Mollari [16] reported finding Aspergillus fumigatus in scrapings from the maxillary sinus of a patient 74 years old. The organism was identified in material fixed in a solution of formaldehyde. The recent report of Adams [17] is of considerable interest.

The data herein reported were collected from studies of material found in a patient who had suffered a good many years with nasal discomfort and discharge.

REPORT OF CASE

The patient was a school teacher, 48 years old. Her mother died of heart trouble at the age of 63 after a long illness. Her father died of cancer. Two maternal aunts died of heart trouble. She had five sisters and one brother. One sister and the brother had heart trouble, and another sister had arthritis. The others were living and well.

As a child, the patient had measles, chickenpox and scarlet fever. She had typhoid pleuropneumonia at the age of 15. She thought she had hay fever as a child and also hives. She had had a more or less chronic nasal catarrh for a long time.

The patient was first seen in April, 1927, when she was suffering from acute rhinitis. At this time a general physical examination gave negative results. The nose and accessory nasal sinuses showed considerable discharge. There was some atrophic change in the nasal membranes. The inferior turbinates had a rather rough and irregular surface. On transillumination the right frontal sinus was somewhat dark, and the right maxillary sinus was dark. The latter was somewhat sensitive. The diagnosis at this time was chronic rhinitis and right maxillary and frontal sinusitis. The patient was under treatment until the middle of June. The acute condition cleared up shortly, but the underlying condition persisted. This condition improved somewhat on the cleansing treatment of the nasal fossae. She was seen in September, 1927, at which time there was some nasal discharge. The membranes seemed dry, and there was little discomfort. The patient had nasal treatment off and on until April, 1928. In October, 1928, treatment was started again and carried on until December, with some improvement. The patient was seen again in May, 1929, at which time the condition was little changed from that first seen. In October, 1930, she was seen twice with acute laryngitis, at which time the sinuses showed no shadow. In December, 1931, she has a supravaginal hysterectomy for displacement. In May, 1932, she was again seen with the nasal condition unimproved. Both maxillary sinuses were irrigated, and a definite infection was found in each. On May 30, she had a radical removal of the left breast for chronic mastitis. At that time the right breast showed some diffuse induration. Treatment of the nasal condition was begun in September, 1932, and regular irrigation of the maxillary sinuses was also instituted. At the time a

15. Mackenzie: Bull. Johns Hopkins. Hosp. **4**:9, 1893.

16. Mollari, M.: Case of Mycosis of the Antrum, Ztschr. f. Hals-, Nasen u. Ohrenh. **25**:65, 1929.

17. Adams, N. F., Jr.: Infection Involving the Ethmoid, Maxillary and Sphenoid Sinuses and the Orbit Due to Aspergillus Fumigatus, Arch. Surg. **26**: 999 (June) 1933.

general physical examination showed nothing unusual. The urinalysis gave negative results. The blood count showed 9,450 white cells, with 41 per cent lymphocytes and 55 per cent polymorphonuclears; otherwise the count was not unusual.

Course of Nasal Treatment.—Beginning April 16, 1927, the following course of treatment was followed: April 16 to June 20, 1927, suction irrigation; Sept. 10 to Oct. 31, 1927, suction irrigation; Nov. 11, 1927, to April 30, 1928, mild silver protein tampons; Oct. 13 to Dec. 15, 1928, mild silver protein tampons; May 13 to 17, 1929, mild silver protein tampons; Oct. 25 to 29, 1930, suction irrigation. Acute laryngitis developed in October. Figure 1 shows the clinical course of the nasal condition beginning May 25, 1932.

TECHNIC

The material was obtained from the maxillary sinus in the following manner:

A local anesthesia was induced by the topical application of cocaine hydrochloride. The nasal cavity was thoroughly cleansed by washing with sterile saline solution. The maxillary sinus was entered with a trocar by puncturing through the naso-antral wall beneath the inferior turbinate. The sinus was then washed out thoroughly with about 250 cc. of autoclaved physiologic solution of sodium chloride which was made up with distilled water and sodium chloride tablets. The returned solution was collected in basins and transferred to sedimentation glasses.

The smears were obtained in two ways. If the pus was in masses so that it could be picked up with a pipet, it was smeared directly. If the pus was in the form of fine shreds or cloudy material, it was centrifugated; the supernatant fluid was decanted, and the residue was smeared and allowed to dry.

Smears were fixed in Downey's[18] fluid. This was done as soon as they were dry. If abundant mucus was present in the pus, the smears dried slowly. In this instance they were allowed to dry until the edges of the material were adherent and then they were placed in the fixative. After the slide had been in Downey's fluid for at least one hour, it was removed, rinsed in tap water and then placed in compound solution of iodine 1:40. After from three to five minutes, the preparation was removed, rinsed in distilled water, and placed in 0.25 per cent solution of sodium thiosulphate for a period of twenty to thirty minutes to remove the iodine. The slide was then rinsed in distilled water and placed in a bath of distilled water for from ten to twenty minutes, when it was removed and allowed to dry.

The preparation was stained with Giemsa's stain for a period of fifteen minutes, after the stain had been diluted 1 drop to 1 cc. of distilled water. It was allowed to differentiate in a bath of distilled water for from three to five minutes. The preparation was dried and mounted in balsam.

ORGANISM

The organism is characterized by the presence of hyphae and spores.

The hyphae are unicellular. They are seen singly and in masses. In the masses, a mycelial-like network is readily made out. This network

18. McClung, C. E.: Handbook of Microscopical Technique, New York, Paul B. Hoeber, Inc., 1929, p. 256.

is brought about by spores and buds attached to one hypha bridging to the next, but the hyphae may be in direct contact with each other, or a slender, finger-like process may extend out to a spore. In each case a definite partition separates one organism from another. Spores may be present around the larger end, along the side or around the smaller end.

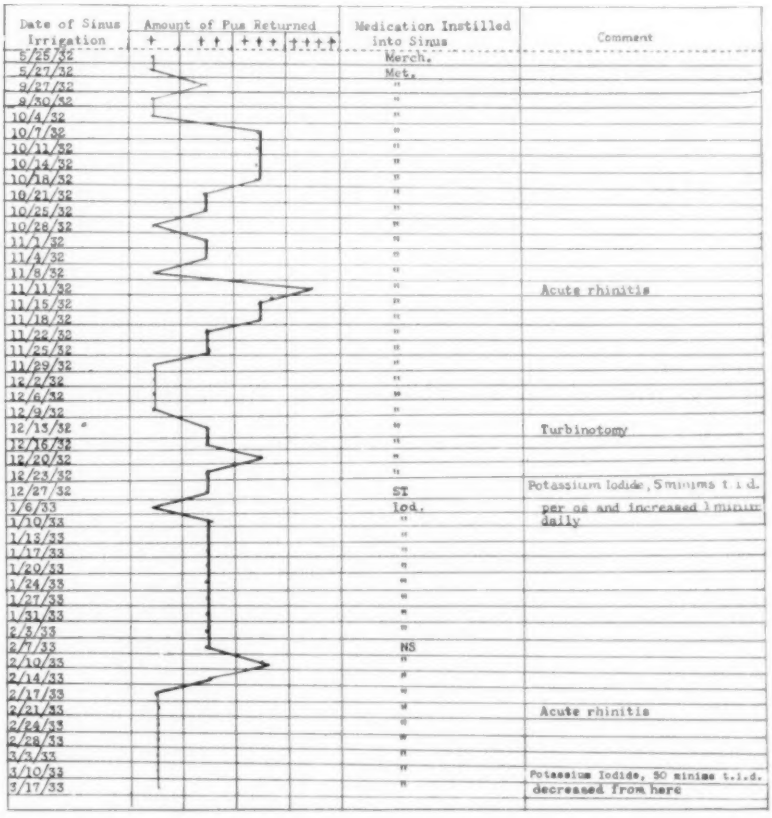

Fig. 1.—Clinical chart showing course of nasal treatment beginning May 25, 1932. *Merch.* indicates mercurochrome, 1:500; *Met.*, metaphen, 1:4,000; *ST*, hexylresorcinol, 1:4,000; *Iod.*, compound solution of iodine, 1:10; *NS*, neosilvol, 10 per cent.

When the hyphae are seen singly, they are club-shaped. Modifications of this are seen in forms which are boat-shaped, and again in forms which are short and comparatively thick, and rarely in forms which are quite long and thin. In fresh preparations they are usually diffusely granular, although a more or less definite nuclear vacuole may be present. In preparations stained by the Giemsa method the hyphae take a uniform rather deep pink stain. Chromatin-like material is usually present but

in a variety of forms and locations. It may be seen at the periphery of the cell as a thin line. It may occur as a diffuse dust at the larger end. It may be present in heavy masses at the larger end, or it may be present as a well defined nuclear mass in the center of the organism.

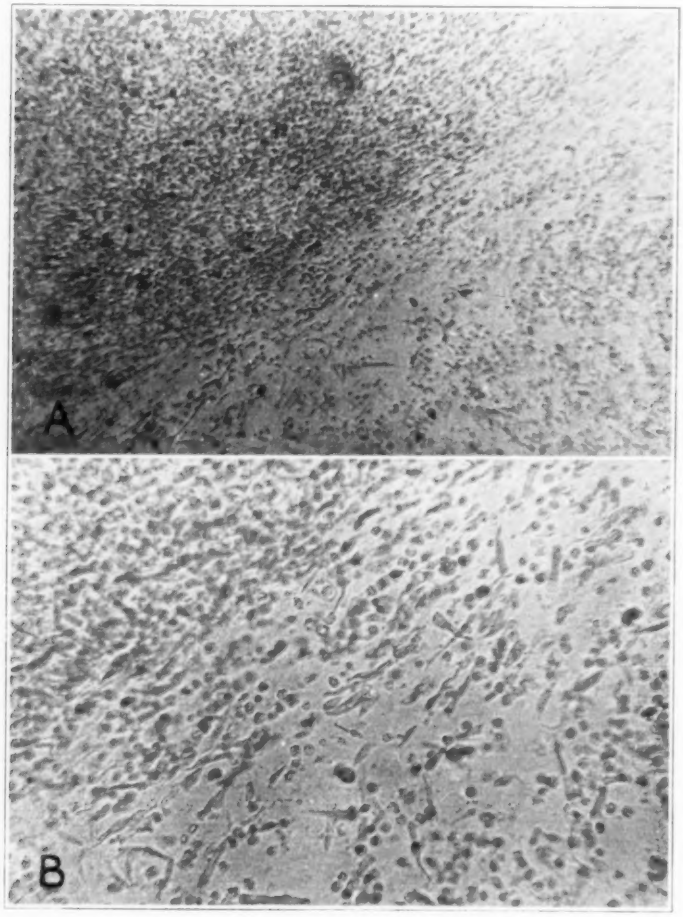

Fig. 2.—*A*, edge of mycelial mass showing individual hyphae and spores. Fresh preparation; × 80. *B*, same as *A*. Fresh preparation; × 160.

The larger end of the hypha presents different appearances. It may be a plain square end. It may be square with a spore in contact. Chromatin may be bunched here in an ill-defined mass or in many small masses as if a large spore, being part of the large end, were dividing into daughter cells. A brush is not infrequently seen at the larger end. This appearance is caused by many delicate finger-like processes extending a short distance out from the square end. It is rather sharply demarcated

from the rest of the organism. The larger end is infrequently seen to look like a socket or cornucopia from which something has been emptied, suggesting the evacuation of a spore or daughter cells. A short delicate finger-like process may grow out from this end.

The hypha is formed by budding out from another mycelium, or it may be formed from a spore by the sending out of a finger-like process. Hyphae are probably formed by the direct growth of daughter cells.

A refractile granule may be seen along the side of the hypha or at the small end, especially when this is forked. A delicate process may extend from the smaller end to some distance away, where it often ends in a spore.

The hypha varies in length from 15 to 40 microns and in width from 4 to 12 microns. The spores vary in diameter from 7 to 15 microns.

Spores arise by budding and by segmentation. Buds are seen on the hyphae and on both kinds of spores. Although spores are often seen attached to the larger end of the hypha, the buds arise from the sides. When the buds contain chromatin, it seems to be derived from along the outer wall of the hypha by inclusion in the spores. A second spore may be seen on the far side of the attached spore, and this may be plain or it may contain chromatin.

Spores are of two kinds, granular and plain. In fresh preparations the spore appears as a granular cell with the nucleus ill-defined from the cytoplasm but often separated into lobules. The surface of the cell gives the impression of irregularity, and occasionally a spore is seen having many fine projections from its surface. The granular spore in stained preparations is seen as a cell with pale pink to clear cytoplasm, and it has a round or lobular, dark blue nucleus which often has bars connecting the lobules, similar in appearance to a polymorphonuclear leukocyte.

The plain spore is nongranular with undifferentiated cytoplasm. In fresh preparations it varies much in size from one quite small to one almost the size of the granular spore. It is often seen to be indented on one side, thus giving the appearance of a saucer. The cell membrane seems to be smooth and somewhat thicker than in the granular spore.

A few fresh preparations showed an occasional granular spore with a brush on one side similar to the brush seen on the hypha. Buds are seen at times on the spores, and sometimes finger-like processes as if hyphae were being formed from them.

Spores are seen in some specimens to form chainlike bodies, several spores in these instances being connected by a short bar or a short cylinder of nonstaining, highly refractile substance. When in clumps, they are usually associated with hyphae.

In some fresh preparations a large cell is seen which is round and two or three times the size of the ordinary spore. It has a large clear area tending to be moon-shaped and occupying nearly a half of the periphery of the cell. The other part of the cell seems to be filled with rather large granules. This probably represents the formation of daughter cells by the spore.

Spores divide, as determined by changes seen in stained specimens, by direct fission and by segmentation into daughter cells. In direct fission,

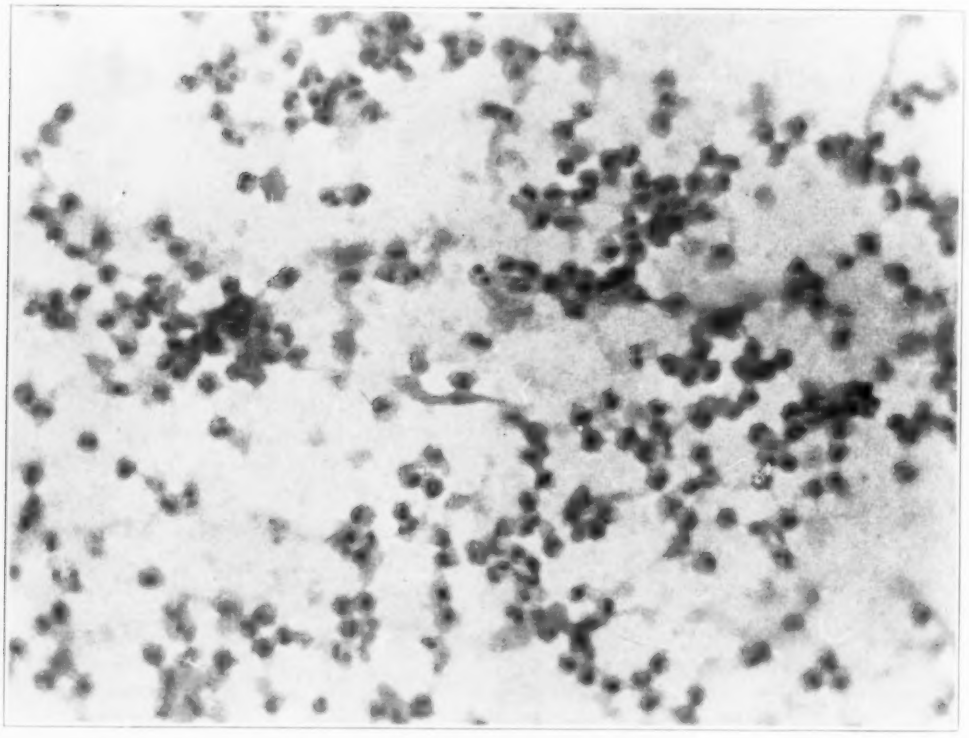

Fig. 3.—Spores and hyphae attached giving mycelial network. The spores show lobulated nuclei resembling polymorphonuclear leukocytes. The hypha in the center has a budding plain spore attached and two spores with lobulated chromatin. Giemsa's stain; × 280.

the cell divides by constriction of the cytoplasm with inclusion of a portion of the chromatin material. The chromatin included is pinched off from the nucleus, or it may be in the form of one of the lobules of the nucleus.

When segmentation takes place, the process starts with a spore having a round nucleus. The nucleus becomes lobulated with as many as six lobules connected by delicate chromatin bars. At times there is a

central mass slightly differentiated from the rest of the cytoplasm by being a deeper pink. The chromatin bars may be seen extending partly around the margin of this mass. The lobules, at first small and uniformly blue, enlarge and become somewhat irregular in outline, and then a pale central area develops in each lobule. As the enlargement con-

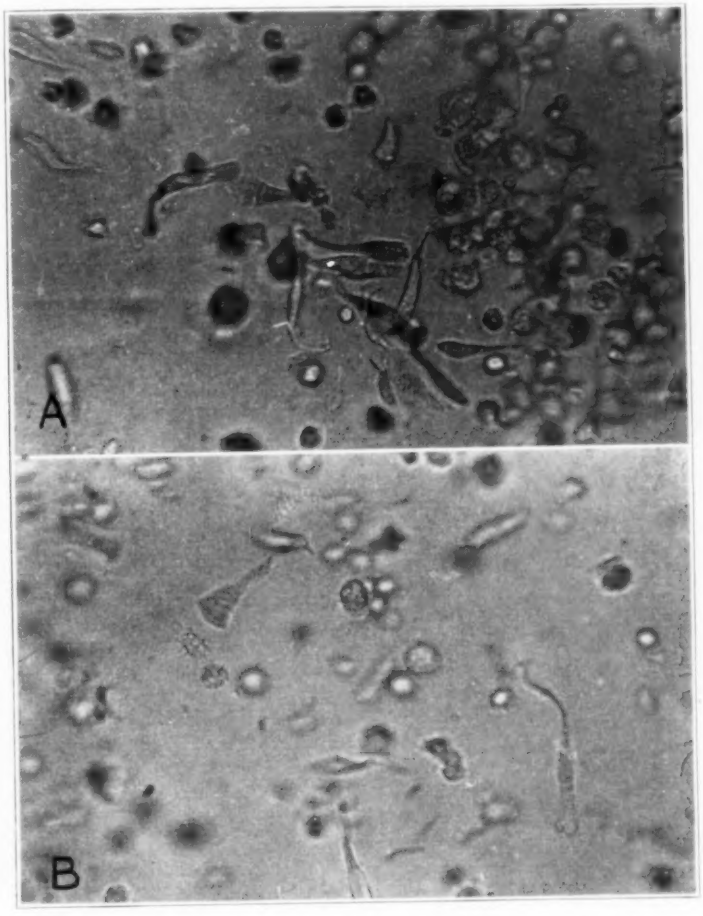

Fig. 4.—*A*, hyphae and spores. One hypha shows brushlike formation on the large end and spores attached. Fresh preparation; × 320. *B*, individual hyphae and spores, one spore with delicate projections. Fresh preparation; × 320.

tinues, the central pale area becomes segmented through the blue mass, and these pale areas are less distinctly seen. This process continues until the lobules are spread out in a fanlike mass. The cell membrane then disappears, and these fanlike masses are seen to lie free, often attaining a size two or more times that of the original spore. From

this point they seem to fade out as if the daughter cells had become disseminated. Occasionally a spore is seen with the cell membrane intact, but it is filled with a blue-staining mass. This mass may be similar in appearance to the fan formation, or it may be more granular, the gran-

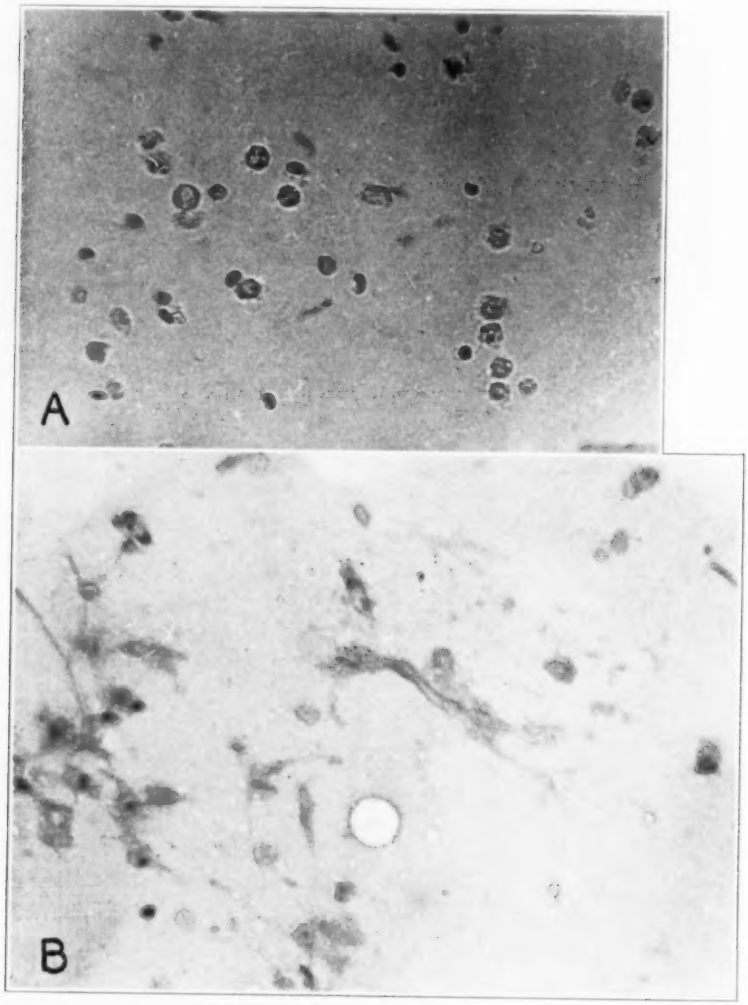

Fig. 5.—*A*, granular spores and plain spores. Fresh preparation; × 320. *B*, atypical hyphae and atypical spores. Giemsa's stain; × 370

ules staining purplish, or the granules may be indefinite and associated with pale areas which are more sharply outlined than usual. The daughter cells are so minute and indefinite that their course after dissemination is not as yet determined.

The morphologic changes noted in the organism during the course of the disease are of considerable interest. As will be noted from the clinical chart, irrigations of the sinus were carried out over a period of months at regular intervals. From these irrigations, smear preparations were made and examined. At first the organism was represented by spores that closely resembled body cells, with the hyphae so atypical and scarce that they were overlooked or classed as débris. This stage is represented clinically by the presence of scanty discharge in the sinus, but there is considerable discomfort with general discharge of mucus from the nasal membranes. In the next stage, which might be termed the middle stage, the discharge in the sinus is greatly increased, but the

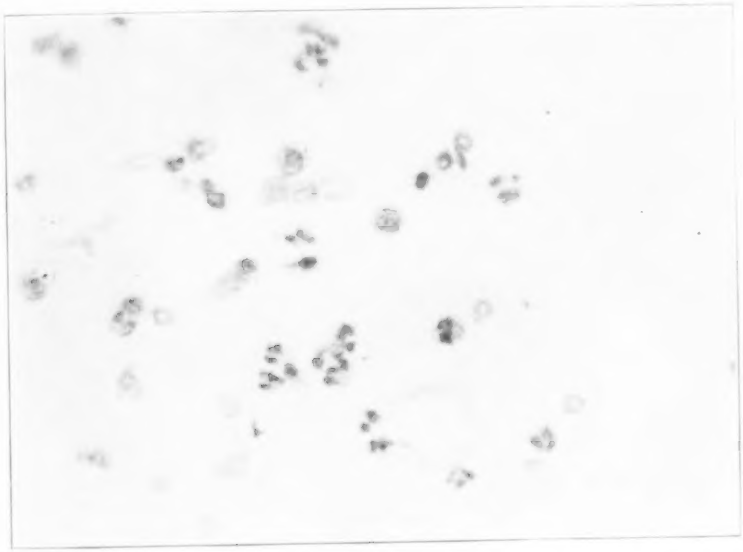

Fig. 6.—Spores with lobulated chromatin showing different early stages of segmentation. Giemsa's stain; × 370.

nasal discomfort is less and the discharge from the nasal mucosa is less. In this stage the hyphae and spores become abundant and form mycelial networks. This stage gradually goes into the third stage in which the discharge in the sinus is scanty and the general discomfort and the discharge from the nasal membranes are greatly decreased. At this time the organism is represented by scanty, often atypical hyphae and spores which are often widely scattered. It is of interest that washings which when examined in the gross show only a few delicate transparent shreds of mucus in microscopic preparations show the presence of the organisms.

In fresh preparations when the organism is treated with compound solution of iodine 1 : 10, a moderate iodine stain results. There is no evidence of a mahogany or blue stain in either the hyphae or the spores.

In fresh preparations that are allowed to stand for from six to eight days, the hyphae gradually disintegrate, so that they are hard to find, but the spores retain their form although often associated with large vacuole-like masses.

Fig. 7.—Spores showing the different forms taken by the chromatin during segmentation: *A*, early stage; *B*, later stage; *C*, fanlike bodies. Drawing made by projection. Giemsa's stain; × 650.

Cultures made by covering a small drop of the pus with plain agar and inverting over a hollow slide show sluggish growth. In one instance material planted in this manner was kept at from 70 to 80 F. (21 to 27 C.) and transplanted after two weeks. It showed well-formed and evidently healthy organisms at the end of eight weeks from the original

plant. Contaminating organisms interfere greatly with these cultures, but they prove the viability of the organism outside the human body.

The reaction of this organism to iodine, indicating the absence of chlorophyll, shows that it is not in the family of Algae, but a fungus. In classifying this fungus, its morphologic characteristics of nonsegmented hyphae and two kinds of spores, and the segmentation of spores into

Fig. 8.—Hanging drop culture, plain agar, 21 days old, showing well formed spores and hyphae extending a little way from the dark culture mass. Fresh preparation; × 120.

daughter cells indicating a sexual cycle, place it in the class of Phycomycetes and order of Mucoraceae. This is based on Castellani's [19] classification of the fungi found in man, which is also used by Jacobson.[20]

19. Castellani, A.: Fungi and Fungus Diseases, Chicago, American Medical Association, 1928.

20. Jacobson, H. P.: Fungus Diseases: A Clinico-Mycological Text, Springfield, Ill., Charles C. Thomas, Publisher, 1932.

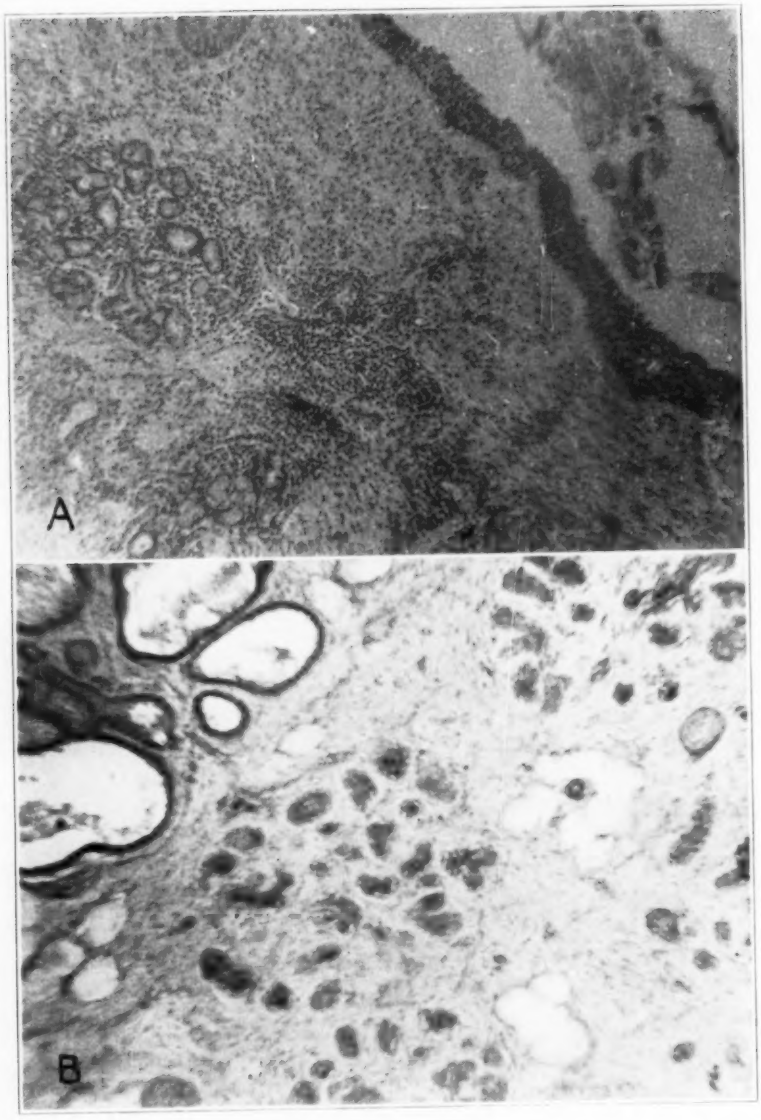

Fig. 9.—*A*, section of turbinate showing epithelium infiltrated with wandering cells and increased connective tissue extending in around the glands with wandering cell infiltration. Hematoxylin and eosin; × 90. *B*, section of breast showing hypertrophied ducts and glands with infiltration of connective tissue and a few wandering cells; × 90. The illustration *B* is made possible through Dr. Francis Edgerton of St. Francis Hospital, New York, who amputated the breast and through whose courtesy I am able to include this photomicrograph.

Search of the literature has so far revealed no fungus which is exactly similar to the one herein described. For convenience, it seems that a name should be given this organism. The resemblance of this organism to body cells is one of its outstanding characteristics, and for this reason the name Mucor-Histoides is proposed for it.

TISSUE

A small piece of the membrane from the right inferior turbinate was removed with a punch for examination. Microscopic study of this tissue in sections showed the high columnar epithelium to be somewhat infiltrated with wandering cells, and in places the goblet cells were increased in size and number, and in other places they were not so numerous. The ducts of some of the glands were somewhat dilated and showed wandering cells in the lumen. There was a decided increase of connective tissue throughout the section with consequent decrease in the prominence of the venous sinuses, and there was a marked infiltration of round cells and polymorphonuclear leukocytes. The increased connective tissue extended in around the gland acini. A diagnosis of chronic inflammatory change in the nasal membrane was made.

Examination of the tissue from the left breast showed typical changes found in chronic mastitis. The uterus showed chronic endometritis associated with a small fibroid tumor.

COMMENT

The repeated finding of the organism herein described in pus recovered from the maxillary sinus indicates beyond doubt that it is a factor in the production of the infection therein, if not altogether responsible for it. How it obtained access to this sinus is not clear, but fungi are so prevalent in nature that aspiration of the organism into the nasal chamber with subsequent extension into the sinus by invasion through the natural orifice seems likely. The presence of a bilateral infection should be noted. It has not been possible so far to discover in the literature another instance of mucormycosis of the maxillary sinuses.

The morphologic characteristics of this organism are of unusual interest because of its resemblance to the wandering cell and even to certain fixed tissue cells. The discovery of the organism was difficult because of this resemblance. Judging by the resistance of the organism to the rather strenuous sinus treatment to which it has been exposed, it must be hardy. The feeble growth obtained in the artificial mediums used indicates that abundant growth takes place only under the most favorable conditions.

The behavior of the organism under the influence of the treatment outlined and the relationship of these changes to the clinical picture are

of considerable interest. At first the spores greatly predominate, the nasal irritation is most marked, and the pus derived from the sinus is scanty. After a few weeks of treatment the hyphae or vegetative portions of the organism become prominent, the nasal irritation is lessened, and the pus obtained is much more abundant. After more weeks of treatment the vegetative portions of the organism become scarce and atypical, the spores less numerous, the pus scanty, and the nasal discomfort and discharge greatly reduced. It is evident from this that the greatest effect of the organism on the patient is exerted at the point of its greatest stagnation and in the presence of scanty discharge within the sinus. The scanty discharge seems to indicate that the effect on the nasal membrane is produced more by a toxin than by the mechanical irritation of the discharge. This toxin may act directly on the nasal membrane, causing it to produce excessive mucus. It may act by absorption into the blood stream and then by action on the nasal membranes through this medium. Another possibility is that it irritates the nerve-endings within the sinus, producing a reflex reaction on the nasal membranes. The clinical improvement as the discharge in the sinus increases and the growth of the organism increases supports the view that a toxin is formed during stagnation. The fact that the amount of pus is scanty when the toxic activity of the organism is greatest suggests the possibility of the organism producing in its period of stagnation some element which inhibits the production of pus. This is rather supported by the fact that the discharge increases after the sinus has been cleansed by the irrigation and the membranes somewhat revived. Another point of interest is that the moisture introduced during irrigation seems not only to increase the activity of the sinus membrane in the production of protective pus but also to increase the growth of the vegetative part of the organism.

The chemical treatment of mucormycosis of the maxillary sinuses in this case has reduced the discharge from the sinuses to faint traces of shreds of mucus in which some of the organisms are present, and the clinical symptoms are greatly relieved. It is difficult to judge, since results were so slowly produced, which of the antiseptics used was the most effective. There is no doubt that the potassium iodide administered by mouth was a factor in reducing the infection. That it should be employed is indicated by its action in this case as well as by its beneficial effect in actinomycosis and other types of mycotic infection. The chemicals instilled into the sinus were the ones generally used in sinus infection, but the results with them were not striking. They were undoubtedly a factor in the improvement of the condition, however, together with the cleansing effect of the sinus irrigation. It is possible that the use of different chemicals from time to time is advantageous in

combating this infection because of the possibility of the organism becoming tolerant if in contact with one agent for a long time.

The chronicity of the infection in this instance is well established by the duration of it while under observation. Experience teaches that in such cases some form of operative drainage of the affected sinuses would be beneficial. The recent major surgical operations which the patient has been subject to, together with the possible necessity of removal of the other breast by radical operation, have made it seem unwise to undertake operative work on the maxillary sinuses, especially since clinical improvement has been so marked.

That the microscopic changes noted in the nasal membranes are due to the mucormycosis described cannot be definitely stated. Judging from the presence of the gross changes in the membranes when the patient was first seen six years ago, and from the fact that it takes considerable time to produce such changes in tissue, the onset must have been quite a long time prior to this. The long period of strenuous treatment, instituted before any great improvement in the infection with this organism could be obtained, indicates how chronic the infection is. It is impossible to prove any connection between the two. The evident long standing of both conditions suggests a relationship. There is little evidence to show that such conditions can be produced by fungi. In this connection, rhinosporidiosis and ozena have already been referred to.

The presence in the same person of a precancerous lesion of the breast associated with this unusual type of fungus infection of the maxillary sinus is of interest. It is impossible to prove that this is anything more than coincidence. Mention should be made in this connection of the monograph of Clarke [21] in which he attempted to prove that cancers and other growths are associated with infection by various fungi.

Clinically and in the gross pathology, mucormycosis does not differ materially from other chronic infections of the maxillary sinus. If routine microscopic examination of pus removed from sinuses is carried on over prolonged periods the chance of detecting this type of infection is increased.

SUMMARY AND CONCLUSIONS

Bilateral infection of the maxillary sinuses with a fungus of the group Phycomycetes, subgroup Mucoraceae, is reported. The fungus was constantly present over a period of seven months.

The organism resembles human body cells so closely that it is difficult to differentiate it from them.

The organism is present in faint traces of discharge.

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21. Clarke, J. J.: Protists and Disease, New York, William Wood & Company, 1923, p. 229.

Morphologic changes in the fungus take place as the environmental conditions change.

The organism grows sluggishly in vitro on artificial mediums.

Mucormycosis of the maxillary sinuses in the case reported was associated with chronic inflammatory changes in the nasal mucosa. A chronic mastitis was present in the breasts. The uterus showed chronic endometritis associated with a small fibroid tumor.

The treatment of the maxillary sinus instituted has given clinical improvement, but so far has not eradicated the disease.

LARYNGEAL NERVES

SURGICAL IMPORTANCE IN RELATION TO THE THYROID ARTERIES, THYROID GLAND AND LARYNX

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SAN FRANCISCO

The variation in the course and direction of the recurrent laryngeal nerve on the right and on the left side may be explained on an embryologic basis. As the heart and the great vessel of the chest, the aorta, descend from their point of origin behind the face, they carry this nerve with them. This is also applicable to the descent of the right subclavian artery. Both of the recurrent nerves have their point of origin in close proximity to the vessels just mentioned. They are carried downward by the descent of these structures during embryonic life. This can be demonstrated in the presence of an anomalous position of these vessels. Such a condition is accompanied by an anomalous location of the respective nerves.[1] From this brief description of the embryology, the variability that the recurrent nerve may assume can be appreciated. Gross deviations are rare; slight modifications are not uncommon. Pemberton and Beaver [2] reported a case in which the right recurrent nerve was found at thyroidectomy to arise from the cervical portion of the vagus on a level with the superior pole of the thyroid gland. Milianitch [3] reported a similar case. The nerve is usually described as supplying all of the intrinsic muscles of the larynx except the cricothyroid. Its importance in phonation and respiration is academic knowledge.

The recurrent nerve, being a branch of the vagus, may be affected in conjunction with the main nerve, as a result of a lesion that is either supranuclear or nuclear or in the main trunk. In addition, it may be involved at any place along its tract, that is, from the parent stem, the vagus, to its ultimate distribution in the larynx. Most of these lesions

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Read before the Section on Laryngology, Otology and Rhinology at the Eighty-Fourth Annual Session of the American Medical Association, Milwaukee, June 15, 1933.

1. Hooper, F. H.: The Anatomy and Physiology of the Recurrent Laryngeal Nerves, New York M. J. **46**:29, 63, 99 and 150, 1887.

2. Pemberton, John, and Beaver, M.: Anomaly of the Right Recurrent Laryngeal Nerve, Surg., Gynec. & Obst. **54**:595 (March) 1932.

3. Milianitch, N.: Note sur une anomalie d'origine des gros troncs nés de la crosse de l'aorte accompagnée d'une anomalie de trajet du nerf récurrent droit, Bull. et mém. Soc. anat. de Paris **94**:230, 1924.

are due to trauma of some kind incurred during surgical procedures on the thyroid gland. Judd, New and Mann [4] demonstrated experimentally the effect of trauma on this nerve. Their conclusions seem to prove that stretching does not impair the function of the nerve. Ligation and

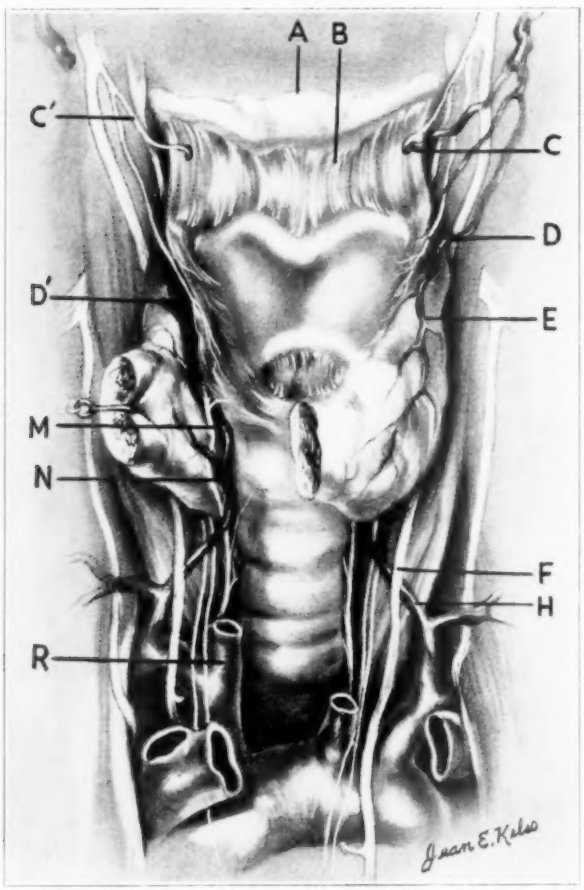

Fig. 1.—Photograph of a drawing of a dissection: *A*, the hyoid bone; *B*, the thyrohyoid membrane; *C* and *C'*, the internal branch of the superior laryngeal nerve; *D* and *D'*, the external branch; *E*, the left superior thyroid artery; *F*, the left vagus; *H*, the left inferior thyroid artery; *M*, the right recurrent nerve, which in this dissection lay anterior to the right inferior thyroid artery at *N*; *R*, the junction of the right carotid and subclavian arteries.

severing, they found, result in permanent paralysis, while pinching causes temporary paralysis of the corresponding portion of the cord.

4. Judd, E. S.; New, G. B., and Mann, F. C.: The Effect of Trauma on the Laryngeal Nerves, Ann. Surg. **67**:257 (March) 1918.

Clinical modification of the voice, without grossly demonstrable lesions in the larynx, permits one to assume that a slight weakness or paresis may occur in such conditions. This assumption is based on clinical observations and the experimental work of Exner, cited by Dilworth.[5] It was hoped that a gross anatomic study might bring out some unknown facts. In addition, the most prevalent location of injury to the nerve during any operative procedure on the thyroid

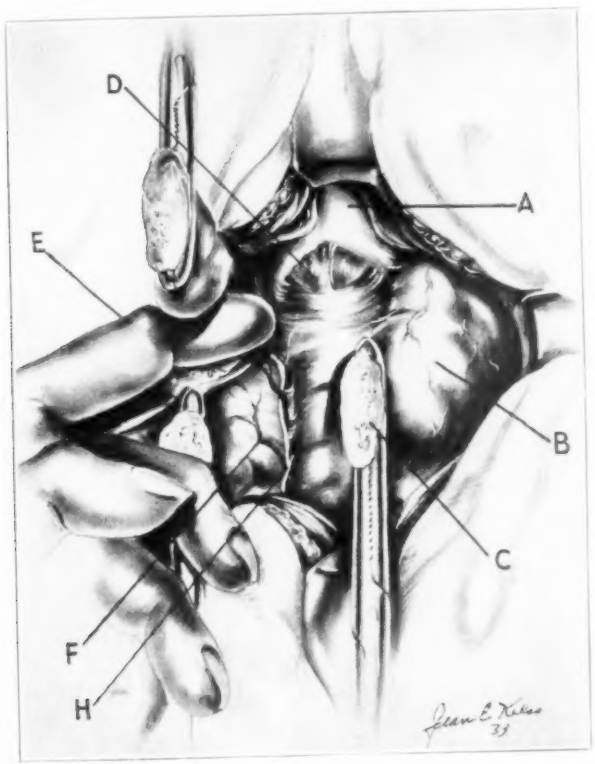

Fig. 2.—Photograph of a drawing of a dissection: *A*, the thyroid cartilage; *B*, the left lobe of the thyroid gland; *C*, the isthmus severed and retracted; *D*, the cricothyroid membrane; *E*, a finger on the inferior cornua of the thyroid cartilage; *F*, one branch of the inferior thyroid artery with the left recurrent nerve posterior at *H*. Just below the index finger may be seen the other branch of the inferior thyroid artery with the nerve anterior.

gland was studied. Forty-two recurrent nerves and fourteen superior laryngeal nerves were dissected, and observations were made as to their anatomy in regard to the thyroid arteries, the thyroid gland and the

5. Dilworth, T. F. M.: The Nerves of the Human Larynx, J. Anat. **56**:48, 1922.

larynx. In addition seven larynges were dissected, and the distribution of the nerves in regard to the muscles of this organ was noted.

Of the forty-two dissections of recurrent laryngeal nerves, twenty-three were on the right side and nineteen on the left. As a general rule, the nerve on the left side was somewhat deeper in the neck than

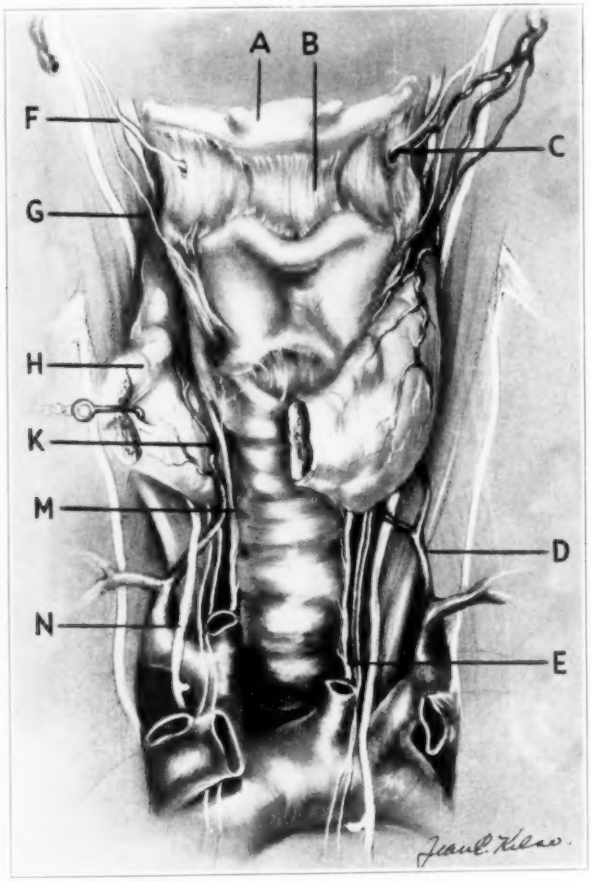

Fig. 3.—Photograph of a drawing of a dissection: *A*, the hyoid bone; *B*, the thyrohyoid membrane; *C*, the internal branch of the superior laryngeal nerve with its accompanying vessel; *D*, the left inferior thyroid artery; *E*, the left recurrent laryngeal nerve; *F*, the right superior laryngeal nerve; *G*, the external branch of this nerve extending to the cricothyroid muscle; *H*, the everted right lobe of the thyroid gland; *K*, the right inferior thyroid artery; *M*, the right recurrent laryngeal nerve posterior to the artery; *N*, the right vagus.

that on the right. It occupied more closely the usual site described in textbooks, the tracheo-esophageal groove. This agrees with the findings of Dilworth.[5] In nine of the twenty-three dissections on the right

side the nerve lay anterior to the inferior thyroid artery (fig. 1). In eight instances the nerve was between the two main divisions of the artery (fig. 2). In six instances the nerve was definitely posterior to the artery (fig. 3). On the left side a somewhat similar condition was observed. Six dissections showed the nerve anterior to the artery; in seven instances it was between the main branches, and in six it was posterior to the artery. This is largely in accord with the findings of

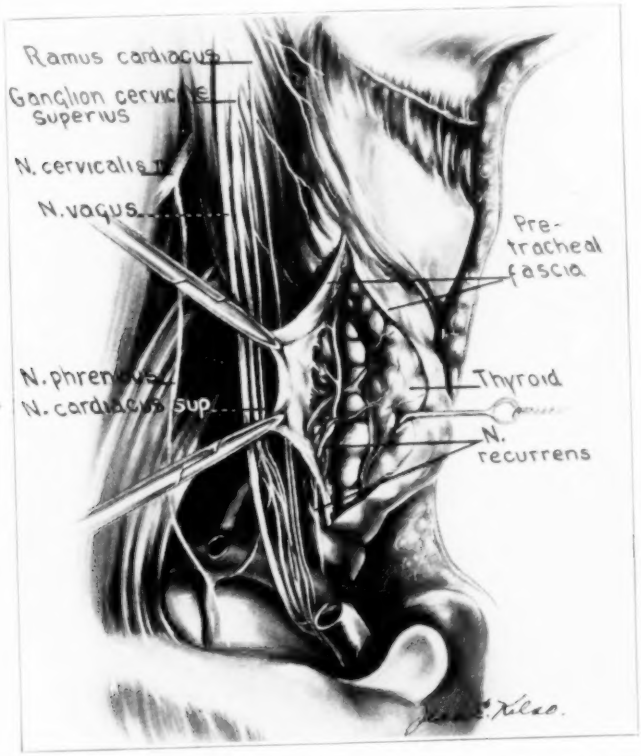

Fig. 4.—Photograph of a drawing of a dissection, showing the capsule of the right lobe of the thyroid gland divided and the relation of the right recurrent laryngeal nerve to the capsule, the gland and the inferior thyroid artery.

Fowler and Hansen[6] and Nordland.[7] It differs somewhat from the observations of Berlin and Lahey,[8] who found a greater disproportion

6. Fowler, C. H., and Hansen, William: Surgical Anatomy of the Thyroid Gland with Special Reference to the Relation of the Recurrent Laryngeal Nerve, Surg., Gynec. & Obst. **49**:59, 1929.

7. Nordland, Martin: The Larynx as Related to Surgery of the Thyroid, Based on Anatomical Study, Surg., Gynec. & Obst. **51**:449 (Oct.) 1930.

8. Berlin, David, and Lahey, Frank H.: Dissection of the Recurrent and Superior Laryngeal Nerves, Surg., Gynec. & Obst. **49**:102 (July) 1929.

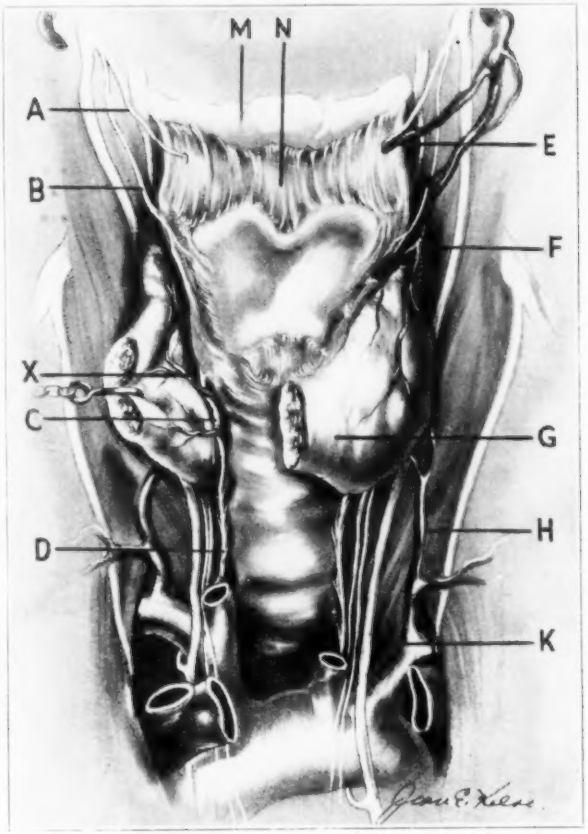

Fig. 5.—Photograph of a drawing of a dissection: *A*, the internal branch of the superior laryngeal nerve; *B*, the external branch supplying the cricothyroid muscle; *C*, the right recurrent nerve anterior to the branches of the right inferior thyroid artery; *D*, the right recurrent nerve; *E*, the left superior laryngeal nerve and vessel; *F*, the left superior thyroid artery; *G*, the left lobe of the thyroid gland; *H*, the left inferior thyroid artery; *K*, the left vagus; *M*, the hyoid bone; *N*, the thyrohyoid membrane; *X*, the right recurrent nerve passing posterior to the right inferior cornua of the thyroid cartilage.

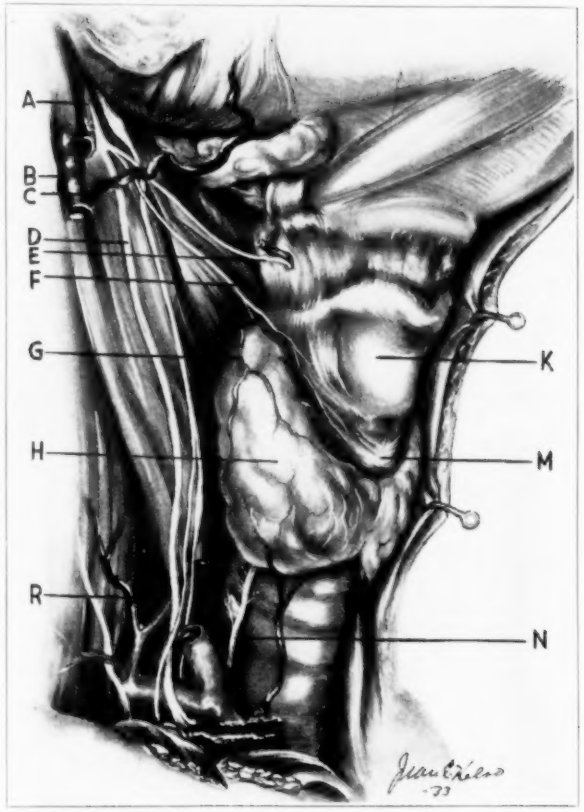

Fig. 6.—Photograph of a drawing of a dissection: *A*, the severed internal carotid artery; *B*, the severed carotid; *C*, the facial artery; *D*, the right vagus somewhat anterior to its normal position; *E*, the internal branch of the right superior laryngeal nerve; *F*, the external branch of the right superior laryngeal nerve supplying the cricothyroid muscle and in relation to the right superior thyroid artery; *G*, the right superior thyroid artery at the upper pole of the thyroid gland; *H*, the right lobe of the thyroid gland; *K*, the right inferior thyroid artery; *R*, the thyroid cartilage; *M*, the cricoid cartilage; *N*, the right recurrent laryngeal nerve.

in the relation of the nerve to the artery on the two sides. Textbooks on anatomy differ as to their description of the course and distribution of this nerve. Gray[9] stated that the nerve may assume a position either anterior or posterior to the inferior thyroid artery.

The relation of the nerve to the capsule of the gland was observed to be different from that described in most textbooks. The capsule is usually mentioned as being formed by the splitting of the second layer

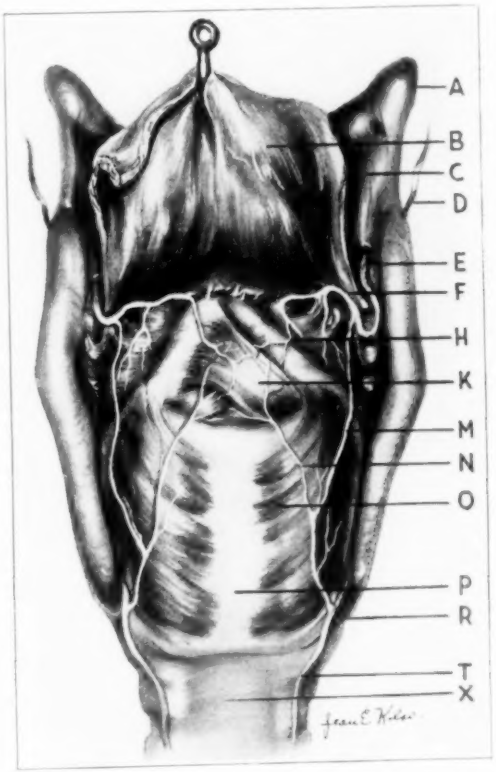

Fig. 7.—Photograph of a drawing of a dissection (posterior view): *A*, the hyoid bone; *B*, the everted phrayngeal mucosa; *C*, the thyroid membrane; *D*, the internal branch of the superior laryngeal nerve; *E*, the internal branch of the superior nerve entering the pyriform sinus; *F* and *H*, a branch of the superior laryngeal nerve extending to the arytenoid muscle; *K*, the interarytenoid muscle; *M*, the communication between the descending branch of the superior and the ascending branch of the inferior laryngeal nerve; *N*, a branch of the inferior laryngeal nerve, commonly known as the recurrent, supplying the arytenoid muscle; *O*, the posterior crico-arytenoid muscle; *P*, the cricoid cartilage; *R*, the inferior cornua of the thyroid cartilage; *T*, the recurrent laryngeal nerve; *X*, the trachea.

9. Gray, Henry: Anatomy of the Human Body, Philadelphia, Lea & Febiger, 1918, p. 914.

of the cervical fascia. Thus the gland is supposed to be surrounded completely by the capsule, and the nerve is accepted as assuming a position at all times posterior to it. This supposed fact was one of the reasons for the development of the intracapsular technic of lobectomy. Moreover, the modern method of ligation of the artery at the capsule was accepted as being devoid of the least danger to the nerve. My observations do not bear this out. This fascial capsule continues for a distance equal to approximately two thirds of the posterior surface

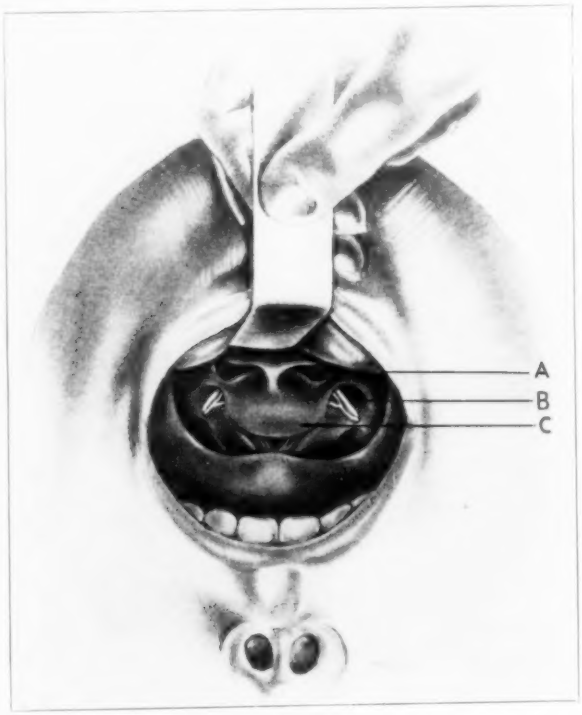

Fig. 8.—Photograph of a drawing of a dissection, showing the internal branch of the superior laryngeal nerve and artery as seen in the modified Rose position: *A* the vallecula; *B*, the internal branch of the superior laryngeal nerve and vessel; *C*, the epiglottis.

of the gland. It is then reflected on the esophagus. A thin modified sheath is continued over the remaining portion of the posterior mesial surface of the respective lobes. The nerve on each side, in practically all of the dissections, was observed to leave this true capsule and occupy a position in close contact with the gland on its inner posterior mesial surface (fig. 4). This makes this portion of the gland the dangerous area in thyroidectomy. It is not difficult to conceive, after studying the anatomy, that inflammatory lesions of the gland proper,

or of its fascial plane, whether from trauma or from other causes, could be readily transferred to the nerve; also that tumors, particularly adenomas and cysts, might have such a distorting influence on the nerve as to cause a prolonged period of traction, with weakness of the fibers and their impulses, sufficient to cause microscopic changes in the innervation of the respective laryngeal muscles, without gross evidence other than clinical manifestations.

The following tabulation shows the anatomic findings with regard to the relation of the recurrent laryngeal nerve to the inferior thyroid artery, the thyroid gland and the thyroid cartilage.

Number	Location of Nerve	Relation to Inferior Thyroid Artery	Relation to Posterior Mesial Surface of Gland	Relation to Inferior Cornua
42	R 23	Anterior..... 9 Posterior..... 6 Between..... 8	Near..... 20 3 (?)	Posterior..... 23
	L 19	Anterior..... 8 Posterior..... 6 Between..... 5	Near..... 17 2 (?)	Posterior..... 19
Total.....	42	Anterior..... 17 Posterior..... 12 Between..... 13	Near..... 37 5 (?)	Posterior..... 42

Fourteen dissections of superior laryngeal nerves were made. The course of the nerve was studied in regard to its two main branches, the external and the internal (fig. 5). In all of the dissections the external branch was readily determined. It occupied a variable position in relation to the superior thyroid artery. In some instances it was in juxtaposition; in others, some distance away. In all of the dissections it was demonstrated as supplying the cricothyroid muscle (fig. 6). The course of the nerve impresses one with the ease with which it may be injured or severed in ligation of the superior artery in continuity. This is particularly true if transfixion is used (fig. 6). It seems that the safest method, so far as limiting the danger to the external branch of the superior laryngeal nerve is concerned, is to ligate the vessel as near the upper pole and the capsule as possible.

The internal branch in all the dissections entered the larynx by way of the thyrohyoid membrane after passing beneath the thyrohyoid muscle. It was always accompanied by the superior laryngeal artery and vein (figs. 1, 3 and 5).

Seven larynges were studied grossly as to the distribution of these nerves. In all of the dissections the recurrent nerve was demonstrated as entering the larynx beneath the inferior constrictor muscle, posterior

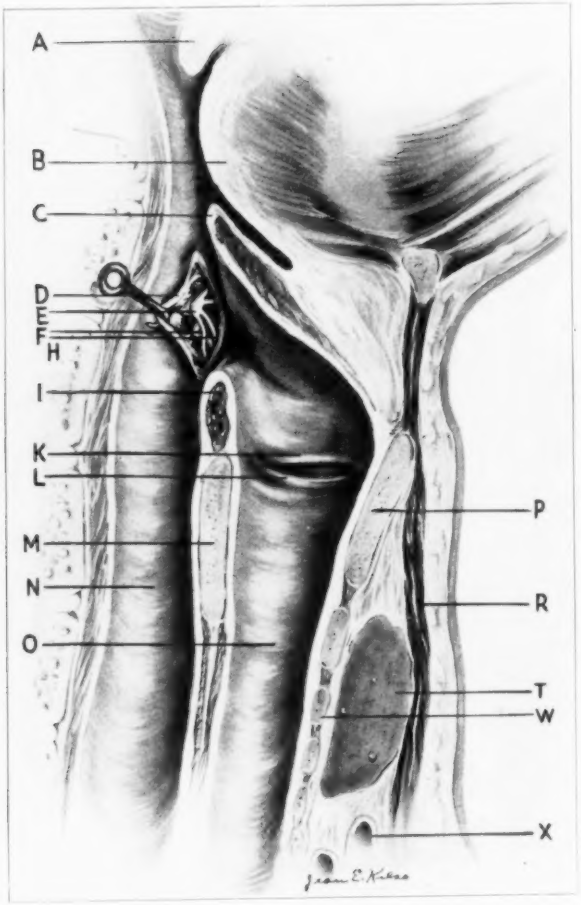

Fig. 9.—Photograph of a drawing of a dissection (sagittal section): *A*, the uvula; *B*, the base of the tongue; *C*, the epiglottis; *D*, the everted mucosa of the pyriform sinus; *E*, the internal branch of the superior laryngeal nerve dividing into three main branches, the upper one redividing and supplying the epiglottis and its immediate region; *F*, the branch to the mucous membrane of the larynx and communicating with the recurrent nerve; *H*, the branch to the mucous membrane and to the interarytenoid muscle; *I*, the interarytenoid muscle; *K*, the false vocal cords; *L*, the true vocal cords; *M*, the cricoid cartilage; *N*, the esophagus; *O*, the trachea; *P*, the thyroid cartilage; *R*, the prelaryngeal muscles; *T*, the thyroid gland; *W*, the tracheal rings; *X*, the vessels of the neck.

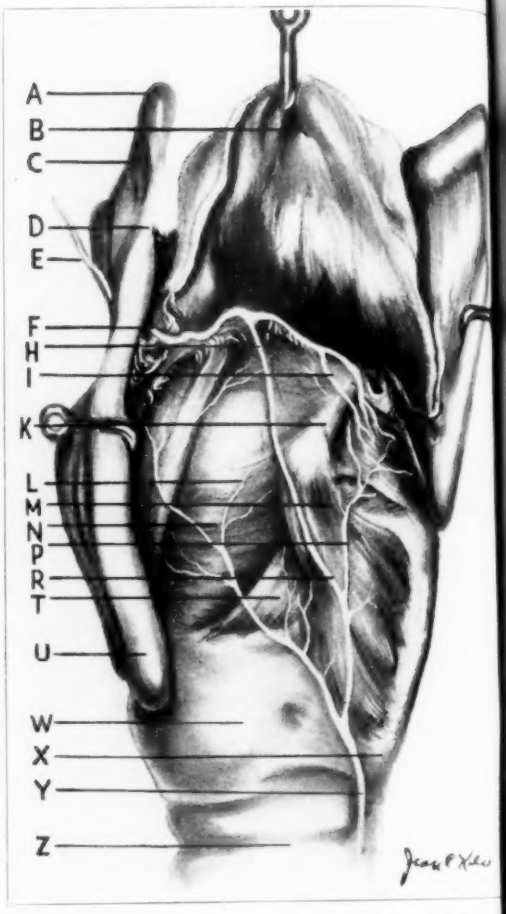

Fig. 10.—Photograph of a drawing of a dissection (posterior lateral view): *A*, the hyoid bone; *B*, the everted pharyngeal mucosa; *C*, the thyrohyoid membrane; *D*, the superior cornua of the thyroid cartilage; *E*, the internal branch of the superior laryngeal nerve; *F*, the nerve entering the pyriform sinus; *H*, the nerve communicating with the recurrent nerve; *I*, the nerve to the arytenoid muscle; *K*, the arytenoid cartilage; *L*, a branch from the recurrent nerve supplying the thyro-arytenoid muscle; *M*, the posterior crico-arytenoid muscle; *N*, the thyro-arytenoid muscle; *P*, the branch of the recurrent nerve supplying the arytenoid muscle; *R*, the communicating branch of the recurrent nerve; *T*, the lateral crico-arytenoid muscle; *U*, the inferior cornua of the thyroid cartilage; *W*, the cricoid cartilage; *X*, the median raphe of the posterior cricoid surface; *Y*, the origin of the posterior crico-arytenoid muscle; *Z*, the recurrent laryngeal nerve; *Z*, the tracheal rings.

to the inferior cornua of the thyroid cartilage[10] (figs. 2 and 5). Fibers could be seen going to the thyro-arytenoid and the lateral and posterior crico-arytenoid muscles as well as to the interarytenoid muscle (fig. 7).

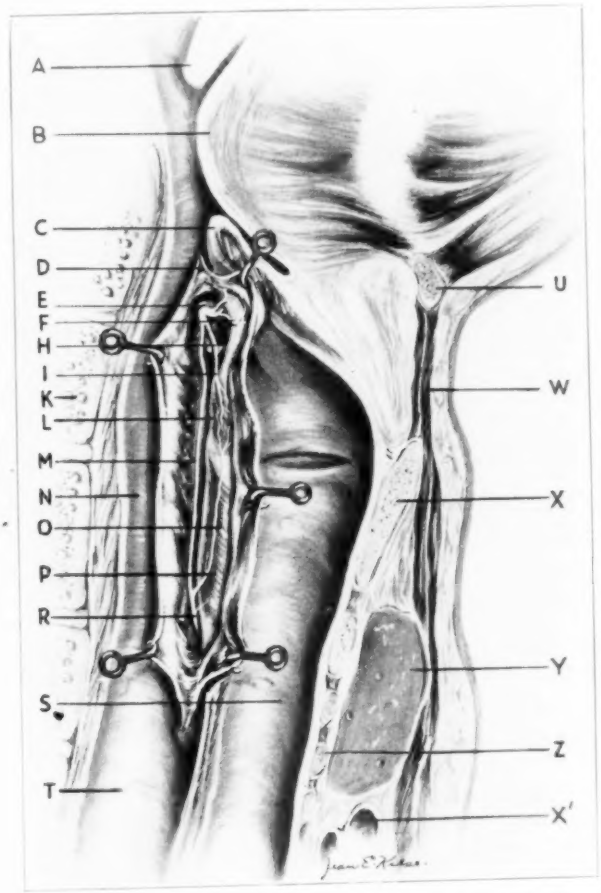

Fig. 11.—Photograph of a drawing of a dissection (sagittal section): *A*, the uvula; *B*, the base of the tongue; *C*, the epiglottis; *D*, the everted mucosa of the pyriform sinus; *E*, the internal branch of the superior laryngeal nerve, with its three main branches of subdivision; *F*, the branch to the epiglottis; *H* and *I*, a branch to the arytenoid muscle; *K*, the vertebra; *L*, the interarytenoid muscle; *M*, the branch of communication with the recurrent nerve; *N*, the lateral pharyngeal wall; *O*, the posterior crico-arytenoid muscle; *P*, the branch from the recurrent nerve supplying the arytenoid muscle; *R*, the recurrent laryngeal nerve; *S*, the trachea; *T*, the esophagus; *U*, the hyoid bone; *W*, the thyrohyoid membrane; *X*, the thyroid cartilage; *Y*, the thyroid gland; *Z*, the tracheal rings; *X'*, the vessels of the neck.

10. Lahey, Frank: Operative Injury to the Recurrent Laryngeal Nerve, S. Clin. North America **12**:839 (June) 1932.

This corresponds with the innervation as described by Negus,[11] Deaver [12] and Jackson and Coates.[13]

The internal branch of the superior laryngeal nerve after entering the thyrohyoid membrane was easily demonstrated beneath a fold of mucous membrane in the sinus pyriformis (fig. 8). It apparently

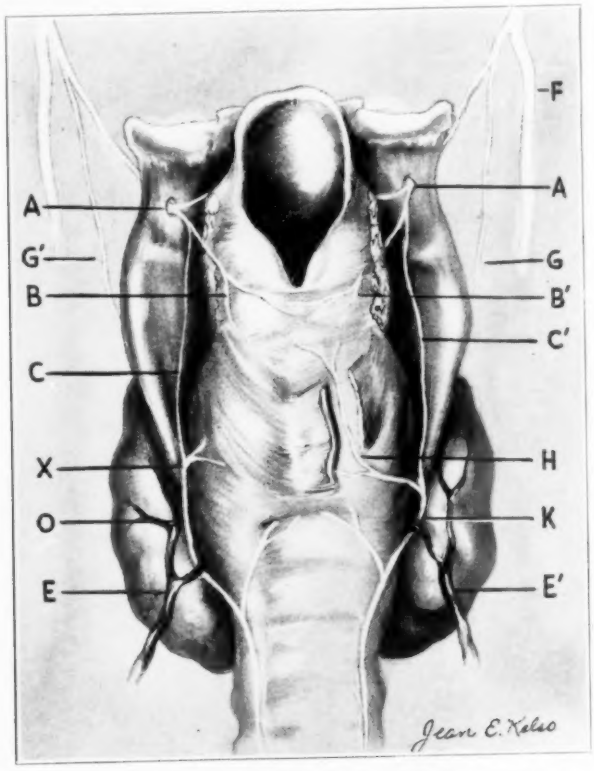

Fig. 12.—Photograph of a drawing of a dissection (posterior view): *A*, the internal branch of the left superior laryngeal nerve entering the pyriform sinus through the thyrohyoid membrane where it divides into three branches; *B* and *B'*, branches extending to the interarytenoid muscle; *C* and *C'*, the descending branch, which communicates with the ascending branch of the recurrent nerve at *O* and *K*; *E* and *E'*, the inferior thyroid arteries; *F*, the vagus; *G* and *G'*, the external branch of the superior laryngeal nerve; *H*, a branch of the left recurrent nerve extending to the interarytenoid and posterior arytenoid muscles; *X*, the left recurrent nerve passing beneath the inferior constrictor muscle.

11. Negus, Victor E.: The Mechanism of the Larynx, St. Louis, C. V. Mosby Company, 1929, p. 462.

12. Deaver, John B.: Surgical Anatomy of the Human Body, Philadelphia, P. Blakiston's Son & Co., 1926, vol. 1, p. 350.

13. Jackson, Chevalier, and Coates, George: The Nose, Throat, and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929, p. 763.

divides into three main branches (fig. 9). These can be demonstrated grossly. One supplies the mucous membrane of the larynx. In six of the seven dissections fibers could be seen extending to the interarytenoid muscle (figs. 7 and 10). The other main branch descended in close relation to the inner surface of the thyroid ala and communicated with an ascending branch of the recurrent laryngeal nerve[14] (figs. 11 and 12).

CONCLUSIONS

1. The recurrent laryngeal nerve may assume a variable position.

2. This is particularly true in its relation to the inferior thyroid artery.

3. This fact impresses one with the wisdom of ligating the artery at some distance from the gland.

4. The nerve apparently leaves the true capsule to assume a position in close relation to the posterior mesial third of the gland proper.

5. This is the dangerous area for injury to the nerve.

6. In all of my dissections the nerve entered the larynx posterior to the inferior cornua, which makes this structure a definite surgical landmark for protection to the nerve.

7. The external branch of the superior laryngeal nerve is in close relation with the superior thyroid artery. It can be injured in ligation of this vessel unless caution is used.

8. The internal branch of the superior laryngeal nerve sends branches which communicate with the recurrent nerve.

9. In most cases it supplies the interarytenoid muscle. This is contrary to the usual teaching.

ABSTRACT OF DISCUSSION

Dr. William A. Kennedy, St. Paul: To what may the accidents to the nerves of the larynx that occur in the course of thyroidectomy be ascribed? Most of the trouble arises from anomalous, unusual or poorly understood relationships between the arteries and nerves. Some investigators point out that the nerve is posterior to the inferior thyroid artery and others, that it is anterior. Lahey stated that injury to the recurrent laryngeal nerve is caused by following the bleeding branches of the inferior thyroid artery down to the trachea. He avoids this by tying the artery extrafascially, securing in addition better hemostasis.

Injury is more likely to occur in those cases in which the nerve pierces the poorly defined capsule and traverses the substance of the gland.

Anomalies of the nerve have been reported, but they are rare and most always associated with anomalies of the aorta or of the right subclavian artery. In the greatest proportion of these cases, the nerve originates from the vagus in the region of the larynx and goes directly to it, while in the other cases it loops around the anomalous vessel.

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14. Morris, Henry: Human Anatomy, edited by C. M. Jackson, Philadelphia, P. Blakiston's Son & Co., 1914, pt. 3, p. 957.

Unusual relationships are more common. Substernal lobes may cause distortion of the recurrent laryngeal nerve out of proportion to their size, and delivering them may cause severe stretching of the nerve. The paralysis may be on the opposite side from the substernal enlargement.

Carcinoma of the thyroid involves the nerves early, and hoarseness may be the first sign to attract attention. In the operation for recurring carcinoma, the danger of injury to the nerves is the greatest because one must feel one's way in order to avoid trouble. Because of the medicolegal aspect all cords should be examined before and after thyroidectomy.

Dr. R. S. Dinsmore, Cleveland: Dr. Ziegelman pointed out that gross deviations are rare, but that slight modifications are not uncommon. That has been my personal observation. I followed the dissections which Dr. Higgins did on a large number of larynges, and his findings coincided rather closely with those of Dr. Ziegelman.

It was interesting to note that the nerve passes anteriorly to the inferior thyroid artery more frequently than posteriorly. It is ordinarily stated in textbooks that the nerve is posterior to the artery.

I agree that the common site for injury to the nerve is not in the tracheo-esophageal groove but slightly lateral to it. In my experience, the three most common sites for injury to the nerve have been: first, at the inferior pole of the thyroid; second, at the terminal branch as it enters the larynx, and third, in close approximation to the tracheo-esophageal groove. I have the least difficulty with injuries to the recurrent laryngeal nerve when I first dissect free the lateral surface of the gland and then place three pilot hemostats. One of these is at the superior pole. I always take care to insert the hemostat from within outward. I have seen the nerve actually caught in the tip of a hemostat passed from the opposite direction. The second hemostat is placed to catch the lateral thyroid vein, and the third at the inferior pole. I have made a special point of placing this hemostat in such a manner that it takes a small portion of the inferior pole with the clamp, placing it so that it stands erect in the wound.

The gland is then dislocated forward, and the dissection from this point can be carried from the trachea outward. After the isthmus has been divided, the hemostats can be placed from within outward in such a manner that they are almost horizontal, and only a small amount of thyroid tissue needs to be left. The points of the hemostats are thus not directed toward the tracheo-esophageal groove.

Dr. David D. Berlin, Boston: In securing the main trunk of the inferior thyroid artery in subtotal thyroidectomy, extrafascial ligation of this vessel is the safest procedure, particularly from the point of view of avoiding injury to the recurrent nerve.

While the recurrent laryngeal nerve usually courses between two fascial envelops, it may pass deep to the external sheath, pierce it, and then occupy a position not only close to but fixed to the posteromedial aspect of the gland. Here one encounters normally an adherent zone just below the arch of the cricoid muscle, representing the area of fixation or attachment of the thyroid gland to the trachea. In this region there exists the most intimate relationship of the recurrent nerve to the gland. The nerve often traverses the strands of glandular and fascial tissue connecting the thyroid gland to the trachea, and thereby comes in close contact with the gland. However, I do not recall ever having seen the nerve completely penetrate the inner capsule of the thyroid and then traverse the entire substance of the gland.

Opportunity to observe this intimate gland-nerve relationship was recently afforded me while I was performing total ablation of the normal thyroid gland in the treatment of chronic heart disease of nonthyrogenous origin, a procedure originally suggested by Dr. Hermann Blumgart, and first instituted at the Beth Israel Hospital in Boston.

Although Dr. Labey and I have reported a greater disproportion in the relationship between the recurrent nerve and the inferior thyroid artery on both sides, otolaryngologists agree that there not infrequently exists a variation between the nerve and artery on either side, and particularly that the nerve may be anterior to the artery on either side. This fact should be borne in mind when one invades the danger zone of the tracheo-esophageal sulcus in the ligation of the inferior thyroid artery, especially in total ablation of the normal thyroid gland.

Dr. Charles H. Frazier, Philadelphia: I have been interested in the dissections of the laryngeal nerve exhibited by Dr. Ziegelman. All surgical treatises deal mostly with the inferior recurrent laryngeal nerve and disregard the superior recurrent nerve; so I shall say little regarding the inferior nerve, except in relation to efforts to promote function in patients with paralysis referred to my associates and me for nerve anastomosis. In those instances we have anastomosed the descendens hypoglossi with the recurrent laryngeal nerve on one or both sides, using the inferior constrictor as our guide to the recurrent laryngeal nerve. I have always disregarded the point at which the nerve is injured, but it is usually at the border of the larynx, near the inferior thyroid artery.

A year ago I turned my attention to the postoperative discomfort in many of these cases resulting from the accumulation of mucus in the larynx. In looking up the anatomy of the superior laryngeal nerve, I became convinced that ligation en masse of the superior pole was impossible without injury not only to the external, but frequently to the internal, branch. Under these circumstances the thyroid gland is enlarged; the superior pole, extending upward, is in closer proximity to the superior laryngeal nerves and therefore it is almost impossible to avoid injury to them. We dissect the superior pole and then isolate the arteries and veins one by one, and ligate each one separately. If one spends enough time and effort, one can readily expose the external branch and see how close it is in its relation to the superior thyroid artery. If one does not isolate the vessels prior to ligation, it is impossible to avoid injury to the nerves. I believe that injury to the superior laryngeal nerve causes this discomfort and accumulation of mucus for from twenty-four to forty-eight hours postoperatively. It may be that the condition of the pharynx causes the mucus to accumulate. Whether that is the mechanism I am not prepared to say, but lately we have tried specifically to avoid injury to the superior nerve, as outlined, and in these cases the patients have had much less discomfort.

Dr. Edward F. Ziegelman: I agree with Dr. Kennedy that stretching of the nerve demonstrates itself by clinical manifestations in the larynx in the form of a change of voice. I have seen it many times in so-called adenomas.

Dr. Dinsmore brought out the interesting point of protecting the external branch of the superior laryngeal nerve. The inferior thyroid artery is surprisingly long, varying from 5 to 12 cm. from its point of origin to its bifurcation at the thyroid gland. It should be ligated away from the gland.

I am inclined to agree with Dr. Berlin in regard to wounding the outer capsule.

The superior laryngeal nerve is interesting to the surgeon. Dr. Frazier brought out the interesting point that the external branch of the superior laryngeal nerve is frequently injured by ligation either at the superior pole or in the continuity.

This external branch supplies the cricothyroid muscle. This is a tensor muscle, and when the nerve is cut or paralyzed hoarseness develops. I have been inclined to believe for some time that the postoperative disturbance when thyroidectomy is performed is not due so much to the recurrent laryngeal nerve as to the external branch of the superior laryngeal nerve. One might ask: Why does the vocal disturbance not continue? There is probably something compensatory in the activity of the other muscles.

A PECULIAR FORM OF HYPERPLASIA OF THE MUCOUS MEMBRANE OF THE UPPER RESPIRATORY TRACT

HENRY BOYLAN ORTON, M.D.
NEWARK, N. J.

Chronic hyperplasia of the pharynx, nasopharynx and larynx, sclerotic hyperplasia of the throat and chronic edema of the throat are synonyms given by Sir St. Clair Thomson [1] in his book on diseases of the nose and throat to designate a peculiar form of hyperplasia of the mucous membrane of the upper respiratory tract. He also states that the etiology is unknown and that the condition is not common.

During the past two years I have had under my observation a patient with a condition that seems to fit this description. Dr. Perry Goldsmith of Toronto has had a similar case, extending over a period of six years, which he has permitted me to cite.

REPORT OF CASES

My patient, M. G., a white woman, aged 32, weighing 119 pounds (53.9 Kg.), was referred to me by Dr. Tirrell on Nov. 20, 1931, because of an unusual condition of the throat. The family history was irrelevant. The patient's general health had been good until the present illness, which began in December, 1930, with a very sore throat, necessitating that she remain in bed for five weeks. This was accompanied by severe pain in the back of the head and some difficulty in swallowing and breathing. The voice was unaffected. At the time of the first examination the patient complained of some soreness in her throat and slight dyspnea. Examination of the nose gave negative findings, and that of the throat revealed a pale hyperplasia of the uvula, the anterior and posterior tonsillar pillars, the epiglottis, the aryepiglottic folds and both arytenoids.

A complete blood count, a Wassermann test, an allergy test, examination of the urine and roentgen and physical examinations were carried out. The urine was normal. The blood count showed: red cells, 4,040,000; white cells, 6,100; polymorphonuclears, 72; lymphocytes, 35; monocytes, 2; eosinophils, 1; blood platelets, 268,400, and hemoglobin, 82 per cent. The sputum showed the presence of epithelial cells, lymphocytes, various cocci, various rods and streptobacilli, Fusiformis and Spirillum. Roentgenograms of the larynx, taken by Dr. Baker, showed marked enlargement of the epiglottis, the shadow of which nevertheless was smooth in outline. The shadow of the ventricle was apparently encroached on from behind. No definite tumor mass could be seen. Stereoscopic examination

Read before the Section on Laryngology, Otology and Rhinology at the Eighty-Fourth Session of the American Medical Association, Milwaukee, June 16, 1933.

1. Thomson, S. C.: Diseases of the Nose and Throat, ed. 3, New York, D. Appleton and Company, 1926.

of the lungs showed no definite pathologic condition. Examination of the skull showed no evidence of abnormalities of contour or density on which to base a diagnosis. The bones and soft tissues of the leg also appeared normal. Physical examination of the lungs gave negative findings. The blood pressure was 120 systolic and 80 diastolic. A sensitization test gave no information.

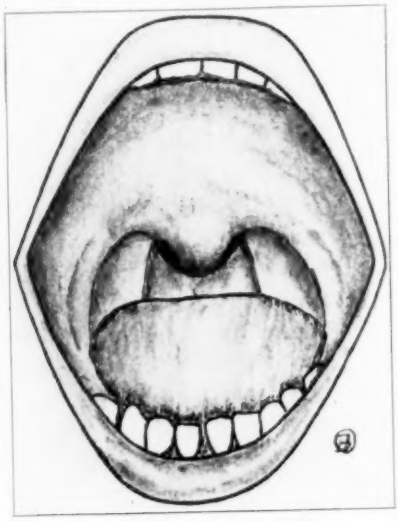

Fig. 1.—Sketch showing the appearance of the uvula and tonsillar pillars. This figure and the following figures illustrate the author's case.

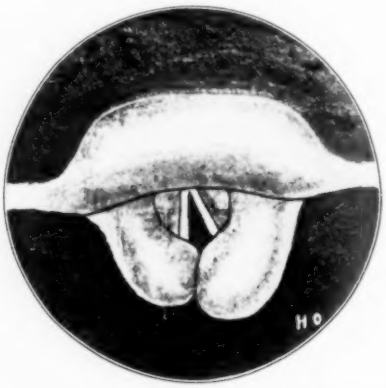

Fig. 2.—Sketch showing the appearance of the epiglottic and arytenoid cartilages.

On Jan. 29, 1932, a small piece of tissue was removed from the junction of the uvula and anterior pillar on the left side. The following report was submitted by the pathologist, Dr. Goldberg: "The deeper layers of the stratified squamous epithelium show mitotic figures in various stages of division. The submucous tissue is edematous, showing numerous, various-sized lymph vessels, some of which

Fig. 3.—Roentgenogram of soft tissue of the larynx, showing the appearance of the arytenoid cartilage and epiglottis.

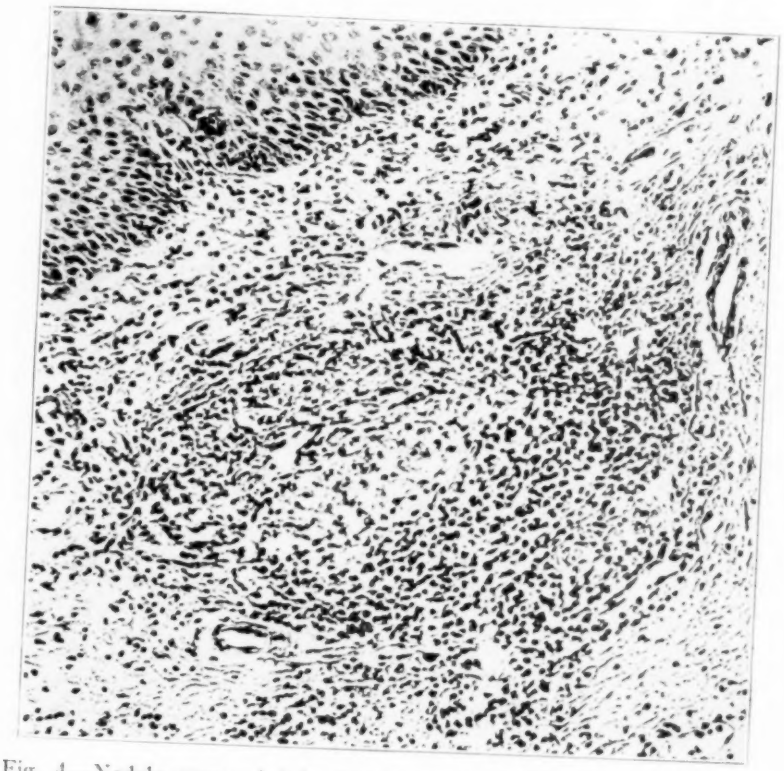

Fig. 4.—Nodule surrounded by lymph vessels and edematous tissue beneath stratified squamous epithelium; × 186.

are filled by a homogeneous substance, while others contain large oval cells with pale-staining nuclei and homogeneous cytoplasm. There are nodular areas of lymphocytes and plasma cells with obliterated lymph vessels in their center. The clear spaces between these nodules contain scattered plasma cells, occasional polymorphonuclear leukocytes and individual small bundles of myelinated nerve fibers. Quite deep in the submucous tissue there are a few glands lined by pseudostratified columnar epithelium showing liquefaction in places. Impression: This appears to be an idiopathic inflammatory reaction characterized by occlusion of lymph

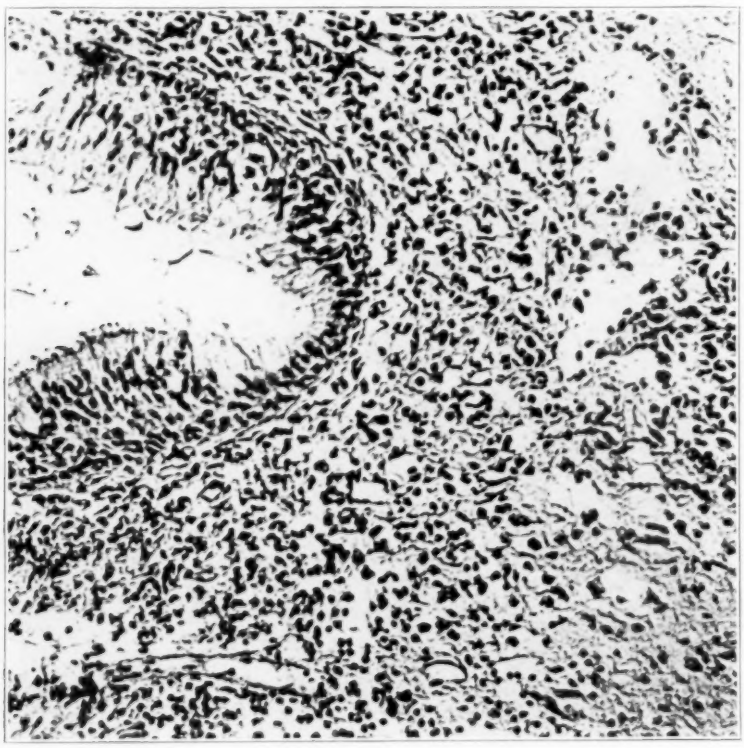

Fig. 5.—Gland lined with columnar epithelium in the areolar tissue; × 250.

vessels with lymphocytic and plasma cell infiltration, the edema being produced by disturbance in the outflow of lymph."

On March 4, 1933, the patient was referred to Dr. Wyatt for high voltage roentgen therapy, and the following dosage was given: Fifteen treatments of the right and left cervical region were given from March 8 to April 5, 1932, with the following factors: 200 kilovolts, 4 milliamperes, through a filter of 0.5 mm. of copper and 2 mm. of aluminum, at a distance of 50 cm. for fifteen minutes. The total dosage was 2,925 roentgens.

Examination of the patient one month later showed a slight improvement of the pharynx. A few weeks later, however, the swelling increased, and the high voltage roentgen therapy was discontinued, as it was feared that tracheotomy might become necessary. The voice was not impaired.

Dr. Goldsmith, of Toronto, furnished the following report of his case:

A. T., a girl, aged 16 years, consulted me in 1924 regarding a nasal cause of attacks of infection of the lower respiratory tract. She had several attacks of pneumonia, and in the previous year had empyema of the chest. The family history and the patient's general health, except for the bronchial condition, were without interest. Examination disclosed chronic pansinusitis on the right side with a similar condition in the left antrum. Pharyngeal and laryngeal examination

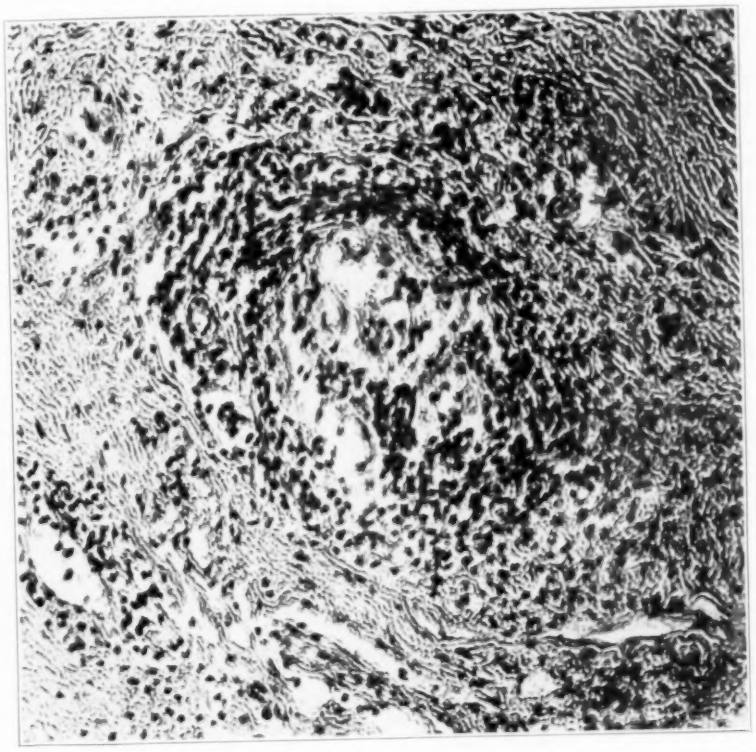

Fig. 6.—Coagulum in lymphatic vessel surrounded by lymphocytes: × 250.

did not show any unusual condition. A suitable intranasal surgical procedure resulted in complete relief of the symptoms.

On Jan. 20, 1927, the patient again consulted me for some discomfort in the lower part of her throat. Her breathing was noticeably noisy. Examination showed marked swelling of the epiglottis and both arytenoid cartilages. There was diffused redness of the lower part of the pharynx. The vocal cords could not be seen; there was no hoarseness, nor did the patient complain of dyspnea. I feared to let her return to her school for the night, but sent her to the hospital, as I feared a spreading of the edema to the larynx.

There was rapid improvement in her condition, but the swelling of the epiglottis and arytenoid cartilage did not lessen greatly; there was only a change from a glistening mucosa to one of a rather waxy color. The swelling has never gone

away, nor has there been any material recession. (Tuberculosis has been definitely excluded—time alone does that now—but injections into guinea-pigs gave negative results). There were localized areas of pale infiltration above the anterior pillars and the soft palate. The Wassermann reaction was negative. Roentgenograms of the chest and examination of the blood showed nothing significant. There was some lymphoid tissue low in the pharynx, extending from the base of each faucial tonsil to a point just above the pyriform sinus. This in itself is far from uncommon and usually is of no importance. On two occasions an acute follicular infection developed, which greatly increased the swelling in the epiglottis and arytenoid

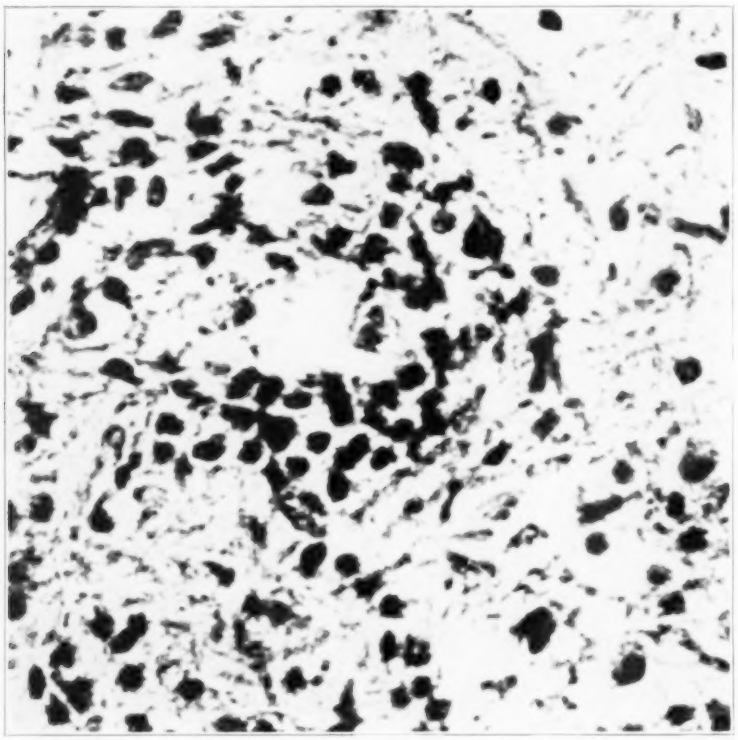

Fig. 7.—Detail of small lymph vessel with perivascular lymphocytic infiltration; × 760.

cartilage. For this reason I removed the remains of the tonsils and the lower lymphoid areas.

During the existence of this peculiar pharyngeal condition (it is not laryngeal but pharyngeal) small lymphoid nodules have developed in various parts of the palate and faucial pillars. Sometimes the palate has a waxy appearance, but there have been no symptoms, nor has the uvula been altered.

Sir James Grant used thyroid extract in a similar case, so I gave this for quite a period. At first the patient thought that she was better, but there was no obvious change. A long course of therapy with mercury preparations was also given, but there was no change. The patient has been observed over a period of

six years and now has no symptoms. Unless a laryngoscopic mirror is used there is no suggestion of any abnormality.

As I see this case, it is one in which, for some reason, marked edema of the lower pharyngeal mucosa has taken place, and this infiltration, except to a small extent, has not disappeared.

REPORTS IN THE LITERATURE

On looking over the literature, I found apparently little available material concerning this condition. In 1901, Brown Kelly [2] reported,

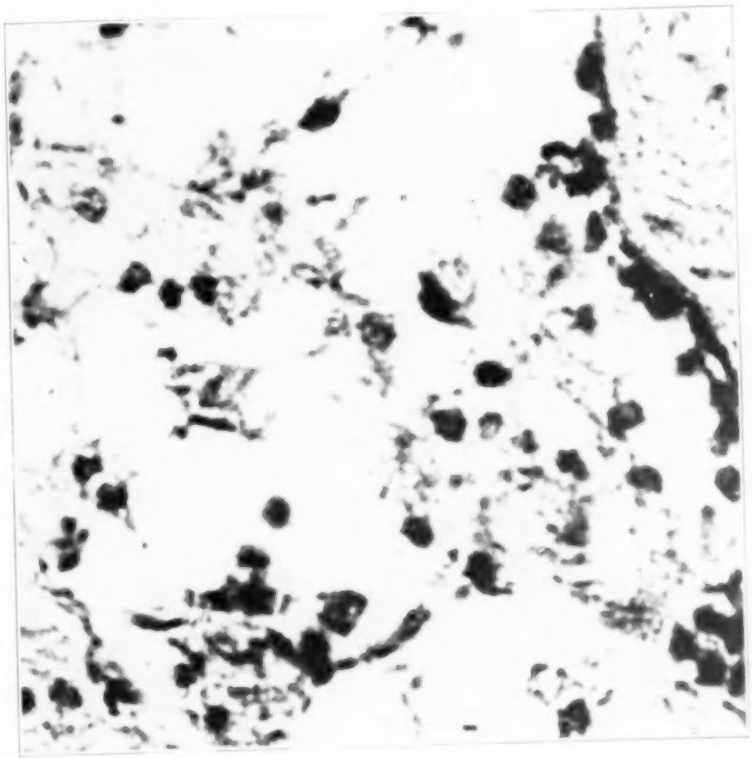

Fig. 8.—Lymph vessel distended with coagulum and disintegrating cells; × 1,000.

under the title "Sclerotic Hyperplasia of the Pharynx and Nasopharynx," the case of a man, aged 34, who had the condition for eight years. A. Logan Turner [3] reported two cases, one of which was presented before the Laryngological Society of London in 1904 and the other before the Scottish Otological and Laryngological Society in 1913,

2. Kelly, A. B.: Lancet **1**:995 (April 6) 1901.

3. Turner, A. L.: Tr. Scottish Otol. & Laryng. Soc., November, 1913; J. Laryng., Rhin. & Otol. **29**:57, 1914.

Sir Felix Semon,[4] in 1905, described two cases of chronic hyperplasia of the mucous membrane of the pharynx and larynx. T. H. Halsted,[5] in 1908, reported the case of a woman, aged 30, under the title "Of a Probable Persistent Angio-Neurotic Edema of the Larynx and Soft Palate, of Three Year's Duration." Gordon B. New,[6] in May, 1933, reported sixty-seven cases of permanent enlargement of the lips and face. Only one patient, however, had involvement of the larynx. In most of Dr. New's patients the condition was associated with facial paralysis.

SUMMARY AND CONCLUSIONS

In reviewing the literature of the other seven reported cases, it is found that the age of the patients at the time of consultation ranged from 16 to 52 years. One patient was 16 years of age; four were in the third decade of life, two in the fourth and one in the fifth; in one report the age of the patient was not given. In the nine cases reported, including Dr. Goldsmith's and my own, two of the patients were males and seven were females. I am unable to account for the greater prevalence of this condition in the female.

The symptoms are fairly uniform; the patient consults a physician for a sore throat, a tickling or a choking feeling in the throat or fulness in the pharynx which he attempts to dislodge.

The general appearance of the patient is very good. There is no hoarseness or cough, and only on examination with the mirror is a smooth, pale swelling of the uvula, the pillars of the fauces, the epiglottis and the arytenoid cartilages discernible. There is no ulceration or interference with phonation. The cause of the condition is not known. There is no hereditary history of a similar condition. The smooth, uniform, more or less diffused permanent infiltration of the uvula and the soft palate, along with that of the faucial pillars, the aryepiglottic folds, the epiglottis and the arytenoid cartilages, and the entire absence of pain or ulceration, with no alteration of the voice, seem to be the outstanding features. In the differential diagnosis rhinoscleroma, tertiary and hereditary syphilis and tuberculosis, as well as Quincke's or, angioneurotic, edema, have been definitely ruled out. Quincke's edema is eliminated by the fact that the condition reported does not disappear. The treatment seems to be entirely symptomatic, and the prognosis is good.

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4. Semon, F.: Two Cases of Chronic Hyperplasia of the Mucous Membrane of the Pharynx and Larynx, Lancet **1**:484 (Feb. 25) 1905.

5. Halsted, T. H.: Tr. Am. Laryng., Rhin. & Otol. Soc., 1908, p. 213.

6. New, G. B.: Tr. Am. Laryng. A., 1933.

The conclusions are as follows: The pharynx and upper respiratory tract may be the site of chronic hyperplasia. The etiology is not known, and the condition is not common; it is unassociated with syphilis, rhinoscleroma, angioneurotic edema or tuberculosis.

ABSTRACT OF DISCUSSION

Dr. Henry M. Goodyear, Cincinnati: The lesion reported by Dr. Orton is unusual. It represents a chronic infiltration of an uncertain nature the cause and pathology of which are alike obscure. The condition is neither edema nor sclerosis. Thomson states that "one case is known to have quite recovered." From reading available case reports it is evident that there is always a primary infection of the throat, "sore throat," or several repeated attacks, with resultant permanent hyperplasia of the uvula, soft palate and pharyngeal tissues, without changes in the voice. Jonathan Hutchinson,[7] in 1893, reported instances of permanent swelling of the tissues of the face following recurring attacks of erysipelas. Dr. William Allen Pusey stated that the cases reported by Hutchinson corresponded to those of permanent facial changes recently reported by New and Kirch (Permanent Enlargement of the Lips and Face, J. A. M. A. **100**:1230 [April 22] 1933).

I believe that these infections may be streptococcic in origin, like cellulitis or erysipelas of the face, resulting in permanent "solid edema" allied to elephantiasis. While the tissues may show an inflammatory reaction early, with occlusion of the lymph vessels and lymphocytic infiltration, later fibrous and inflammatory changes; with chronic cellulitis, would be expected regardless of treatment.

Dr. Ira Frank, Chicago: Chronic edema of unknown origin affecting the throat is likewise described as sclerotic hyperplasia. Chronic edema of the larynx may be a sequel to some of the acute exanthems, especially in children. In adults it may be associated with a chronic condition of the larynx, such as that resulting from tuberculosis, rhinoscleroma or amyloid degeneration of the larynx. It is rarely the result of simple chronic laryngitis. It is also observed in septic infection of dental origin, which may be associated with cellulitis of the face and mucous membranes. It may occur in Bright's disease, syphilis, tuberculosis and iodism. The laryngeal edema of nephritis may represent a terminal stage of the disease, though, on the other hand, it may be a slowly progressive process and occasionally may be the first symptom of unrecognized nephritis. The laryngeal edema of syphilis is usually located about the gummas. Similarly, in tuberculosis the edema usually surrounds the tuberculous ulcer. In idiopathic laryngeal edema the mucous membrane is pale.

Iodine and its preparations may produce chronic edema of the larynx. Some patients are more susceptible than others. In such patients aphonia or raucous voice develops. Laryngeal edema resulting from the use of iodine may be produced or intensified in patients with a previous laryngeal lesion. This is particularly true of syphilitic ulcers which are being treated with iodides.

Tracheotomy is urgently indicated when laryngeal stenosis causes marked dyspnea. It gives relief, though it does not cure the edema.

In a case presented through the courtesy of Dr. Robert Sonnenschein, a woman, aged 28, had her tonsils removed two years ago. She complained of pain in her throat. The sensitization tests for protein showed a positive reaction to several

7. Hutchinson, J.: On the Causes and Nature of Erysipelas, Arch. Surg., London **5**:345, 1893-1894.

vegetables and to fish. A small piece of tissue was removed from the epiglottis for histologic examination. No tuberculous or other specific lesion was found. Edema of both arytenoid cartilages near the edge of the epiglottis has continued, though the patient is not conscious of the swelling. She is apparently in perfect health and is free from distress.

Dr. John J. Shea, Memphis, Tenn.: Dr. Orton has reported an unusual type of disease involving the throat. I saw the patient described by Dr. Goldsmith, and I have seen another patient who was presented by Dr. Robin Harris before the American Laryngological, Rhinological and Otological Society in 1930, under the title "Granuloma Venereum: General Discussion with Report of a Case of Laryngeal Involvement." These two cases had a peculiar clinical manifestation in common. The laryngeal picture in both patients was that of a swollen larynx without ulcerations, the swelling being so extensive as to obliterate the landmarks of a normal larynx. The lymphoid tissue of the pharynx was hypertrophied in each case. Dr. Harris' patient eventually had ulcerations at the base of the tonsillar pillar and on the margin of the epiglottis, and tracheotomy was required.

The histopathologic study of the specimen of tissue taken from the left arytenoid cartilage and the base of the epiglottis contained Donovan bodies and moderate hyperplasia of the mucosa in some areas and ulcerations in others.

The submucosa showed infiltration by polymorphonuclears and round cells. There were marked endothelial proliferation of the lining of the blood vessels and many new blood vessels, together with an occasional vein filled with polymorphonuclear cells. These observations are in contrast to those in Dr. Orton's case, in which the lymph vessels were occluded with lymphocytic and plasma cells, causing an overflow of lymph.

Dr. Henry Boylan Orton, Newark, N. J.: I presented this case before the section with the idea of learning something as to the cause of the condition. Dr. Goodyear referred to Dr. Hutchinson's statement of forty years ago to the effect that edema disappeared to a certain extent and then recurred. In the cases that I have reported the condition did not return to normal at any time. Dr. Frank spoke of iodism. This patient took iodine for a few months, but it did not produce any change in the condition. In the case mentioned by Dr. Shea the condition probably cleared up under specific treatment.

Clinical Notes

ASPIRATING CATHETER FOR THE TREATMENT OF PULMONARY SUPPURATION

NOAH FOX, M.D., AND JOHN W. HARNED, M.D., CHICAGO

The use of bronchial aspiration is widely accepted as an adjuvant to the existing methods of treating suppuration of the lung. This means of drainage is especially important when fluid is present in the parenchyma of the lung in communication with the bronchi. The success of bronchial aspiration is attested by the results obtained at the Jackson Clinic, where Clerf reported thirty-eight recoveries in a possible seventy-six cases of pulmonary abscess. This method of treatment is especially valuable in cases of early abscess, before the normal propulsive function of the bronchial cilia has been disturbed, though even in cases of chronic abscess the mechanical aid that aspiration affords does much toward healing the wound. In cases of chronic abscess, it is known that the normal propulsive function of the bronchial cilia has been disturbed so that they are of little aid in clearing the lung of secretions. It is this mechanical aid that aspiration favors, for it removes materials which are too thick or viscid to be propelled by the ordinary mechanism of cough.

For the most part, evacuation has been accomplished by endoscopic methods, though several observers have blindly inserted catheters perorally, draining the main bronchi, much as the obstetrician aspirates the fluid from the bronchial tree of the new-born.

The bronchoscope is valuable both as a diagnostic medium and for aspiration in suppurative diseases of the lung. It is also likely that its ability to dilate the bronchi is of some value. However, its failure to penetrate beyond the bronchi of the first or second degree results in a distinct disadvantage. Other disadvantages of the bronchoscope must be mentioned. The bulkiness of the instrument makes it so exceedingly uncomfortable for the patient that he fears the procedure, especially when it must be repeated frequently. In his report, Clerf[1] stated that the average number of treatments in each case amounted to sixteen. The economic factor must also be considered, for the patient must be hospitalized and the doctor paid in accordance with the surgical technic involved in the operation. In many sections conditions are not favorable for the procedure, nor are there physicians trained well enough to carry it out. Other disadvantages which may be mentioned are trauma to the bronchial tree and inability to come in contact with the infected area directly for injection of radiopaque material, medication or aspiration.

On the other hand, catheterization by the method to be described obviates most of these disadvantages. We feel that any method that facilitates aspiration should be recorded.

The catheter used with success in our clinic for the past three years is the ordinary soft French catheter with curved olivary tip (fig. 1). After cocainization

From the Department of Otolaryngology, Section of Allergy, University of Illinois College of Medicine.

1. Clerf, L. H.: Pulmonary Abscess Following Tonsillectomy, Arch. Otolaryng. **11**:192 (Feb.) 1930.

sizes from 12 to 16 are readily accepted by the patient without discomfort. The catheter is lengthened by vulcanizing an additional 12 inches (30.5 cm.) to it so that it can reach any remote corner of the chest. The natural curve of the solid tip of the catheter may be made permanent by inserting an ordinary pin into the distal end and anchoring it firmly in the wall of the catheter. This also adds additional opacity to the tip. If the curve is not exactly as required, the wired tip may be bent to the curvature desired. There is enough lead in the rubber of these catheters to cause them to be radiopaque and easily followed under the fluoroscopic screen.

The technic of insertion is as follows. Several cubic centimeters of 5 per cent cocaine is dropped into the nostril and allowed to run down into the pharynx, the patient being warned against swallowing the material. After a lapse of five minutes, a few drops of 10 per cent cocaine are applied to the cords by a tampon.

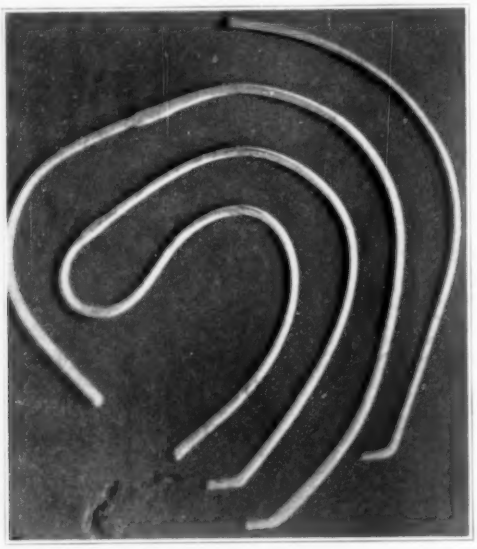

Fig. 1.—The catheter is the ordinary soft French one with a curved olivary tip.

After waiting for another five minutes, 5 cc. of a 2 per cent solution of cocaine is distributed through the cords to the trachea and the main bronchi. The catheter is fed through the nose, past the epiglottis, into the larynx. The curve of the tip is controlled by manipulation of the nasal portion of the catheter. As it passes the tip of the epiglottis under the guidance of the laryngeal mirror, it is rotated and allowed to enter between the cords. The cords must be thoroughly cocainized to allow this. After passage into the trachea, the patient is placed in front of the fluoroscopic screen, and by rotation of the nasal portion of the catheter, it is gradually fished into the area desired. With a small amount of practice, one can quickly pick out the bronchus one desires to enter and learn to turn away the tip from bronchi which are not to be entered. After deftness has been gained in manipulating the tube, the upper lobes may be as easily entered as the lower. The catheter is then attached to the ordinary suction pump, and aspiration is accomplished. If one desires, one can irrigate and aspirate quickly without flooding other areas, or medication of any desired type can be carried out. In

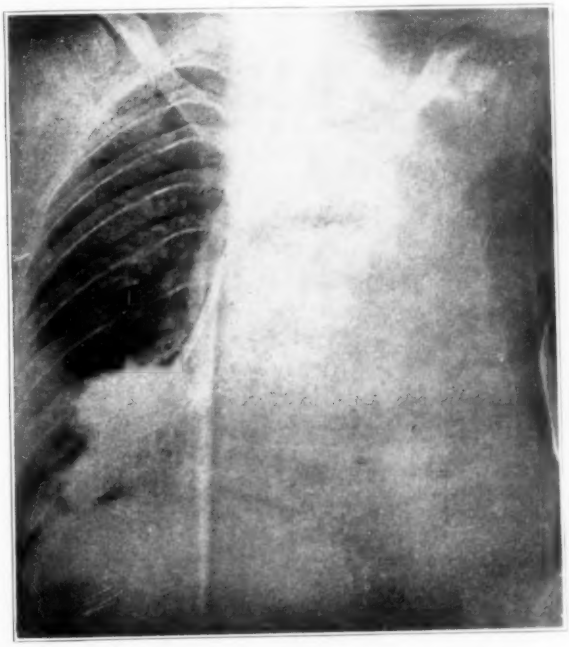

Fig. 2.—The condition of the chest when treatment was begun.

Fig. 3.—The condition of the chest after the tenth aspiration.

addition, the catheter affords an excellent method of placing radiopaque materials in selected areas. If a fluoroscope is available, the entire procedure may be carried out in the office and the patient dismissed with practically no discomfort. A series of pictures taken in a case of multiple pulmonary abscesses occurring after bronchopneumonia is included. The patient had had a fetid bronchitis since early childhood. In 1923, a bronchoscopic examination was performed at Rochester, Minn., and a diagnosis of congenital bronchiectasis was made. There was apparently no further treatment of the condition until about one year before the present writing, when pneumonia developed which was slow in resolution. Examination by a competent internist at that time revealed multiple pulmonary abscesses. Further roentgen study with radiopaque oil revealed the outlines of an abscess in the upper left lobe, another abscess in the lower portion of this lobe and still another in the

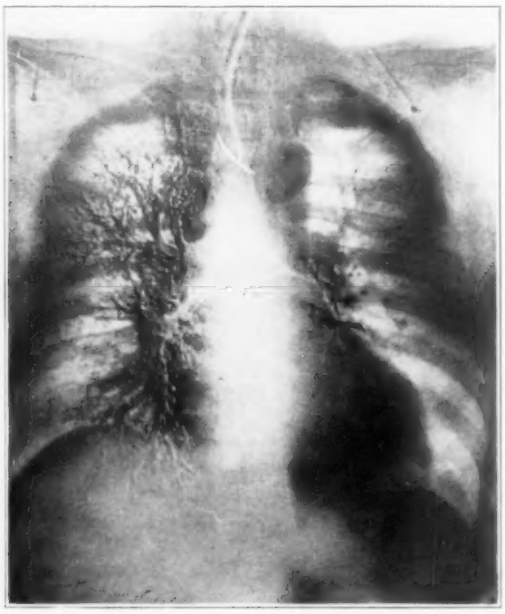

Fig. 4.—Injection of lipoid by means of a catheter.

lowermost portion of the lower left lobe. Because of the condition of the patient and the multiplicity of the lesions, operation was contraindicated. The thickened pleura mitigated against any possible benefit from pneumothorax or phrenicotomy. The patient was referred for aspiration ten months after the onset of the condition. Figure 2 represents the condition of the chest when treatment was begun. Figure 3 represents the condition after the tenth aspiration.

The treatment consisted in aspiration and lavage with physiologic solution of sodium chloride every five days. An antivirus was made from the aspirated material, and after each aspiration and lavage, from 5 to 10 cc. was instilled into the abscess cavities. By intradermal vaccination, it was first ascertained whether or not the patient was sensitive to the material to be instilled, since the organisms were grown on a protein medium. At the time of writing, the patient is free from fever and expectorates only a small amount of odorless mucoid material. She has gained weight and carries on her ordinary household duties. Although we did

not examine her previous to her attack of pneumonia, she assures us that the amount of expectoration now present is less than that which occurred previous to her former illness.

The part played by the antivirus cannot be evaluated from this one case. However, we have a number of other patients with pulmonary abscess and bronchiectasis under treatment, and a report of these will be forthcoming in the near future.

SUMMARY

The catheter, of course, cannot replace the bronchoscope, but when used as a supplement to it the catheter presents certain advantages which may be summarized as follows:

1. The catheter will easily go "around corners" and aspirate an abscess directly, a property lacking in the rigid bronchoscope.

2. The ease with which the catheter can be manipulated does not necessitate a great amount of skill or training.

3. There is a minimum of discomfort and trauma for the patient, which eliminates his fear of the procedure.

4. The procedure obviates hospitalizing the patient, since it can easily be carried out in the office or the fluoroscopic room.

5. The apparatus is exceedingly cheap in cost and upkeep.

6. It allows more accurate localization of pathologic areas with a minimum amount of necessary radiopaque material.

7. It affords a simple method of withdrawing material directly from an abscess for laboratory study.

8. It affords a direct method for lavage of cavities or for any type of medication desired.

9. It provides accessibility to passages beyond the secondary bronchi.

10. As a nonsurgical procedure, it takes its place along with rest, postural drainage and treatment with arsenicals.

NOTE.—Dr. H. L. Stitt, of Cincinnati, uses a similar catheter, although his mode of application differs slightly.

News and Comment

CLEVELAND SESSION OF AMERICAN MEDICAL ASSOCIATION

Application blanks are now available for space in the Scientific Exhibit at the Cleveland Session of the American Medical Association, June 11 to 15, 1934. The Committee on Scientific Exhibit requires that all applicants fill out the regular application form and requests that this be done as early as convenient. The final date for filing applications is Feb. 26, 1934. Any person desiring to receive an application blank should address a request to the Director, Scientific Exhibit, American Medical Association, 535 North Dearborn Street, Chicago, Ill.

AMERICAN BOARD OF OTOLARYNGOLOGY

An examination was held in Boston, Sept. 16, 1933, during the meeting of the American Academy of Ophthalmology and Otolaryngology. Seventy-nine candidates were examined, of whom thirteen were conditioned or failed.

The board will hold an examination in Cleveland, on June 11, 1934, during the meeting of the American Medical Association. Also, early in the summer (the date has not been set) an examination will be held in Butte, Mont., in connection with the meeting of the Pacific Coast Oto-Ophthalmological Society. Prospective applicants for certificate may address the secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, for proper application blanks.

CORRECTION

In the abstract of the article by Dr. W. Brock and Dr. O. Gagel, entitled, "Multiple Sclerosis Mistakenly Diagnosed as Tumor of the Right Cerebellopontile Angle: Report of a Case," which appeared in the November issue (ARCH. OTOLARYNG. **18**:671, 1933), the word "repeated" was omitted from the fifth line from the bottom. This sentence should read: "Possibly in the case reported repeated cochlear and vestibular examinations would have resulted in the correct diagnosis," etc.

Abstracts from Current Literature

Ear

Otitis Media as an Aetiological Factor in Gastro-Enteritis. Leonard Findlay, Arch. Dis. Childhood **7**:307 (Dec.) 1932.

Otitis media has been stressed as a factor in the etiology of gastro-enteritis. The author feels that otitis is not the important factor, but that it may develop during the course of gastro-enteritis and increase the gravity of the case. Bronchopneumonia or pyelonephritis also may develop during the course of nutritional diseases, but gastro-enteritis is the major problem and demands first consideration.

Kelly, Milwaukee. [Am. J. Dis. Child.]

Temperature Curve in Acute Otitis Media in a Child. Liselotte Meyer, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:77 (May 17) 1933.

Meyer reports the results of a study of the temperature curves in 200 cases of acute otitis media in children under 14 years of age, and compares them with those obtained by Schiessl in a similar study of 250 adults at the same clinic. Afebrile cases of acute otitis media occur less frequently in children than in adults; subfebrile cases are more frequent, but the majority of the cases are febrile. Pure cases of otitis are in the minority; usually there are primary or concurrent diseases. In nurslings the aural symptoms may be obscured by severe general symptoms. Bilateral otitis is not always accompanied by a higher average temperature, as it is in adults; it may evolve with an insignificant rise in temperature. The general course of the fever in infantile otitis is distinguished by absolute irregularity; light cases may show a high fever and severe cases, slight fever. Frequently deflections in the fever curve have no apparent cause; the individual power of reaction of the organism seems to be the determining factor. An initial high temperature, 39 C. (102.2 F.), in favorable cases shows a lytic drop in a few days; it is usually of a remittent type. A high continuous fever, from 40 to 41 C. (104 to 105.8 F.), or a fever of long duration points to another disease or a complication; likewise a too rapidly lowered or a subnormal temperature is an unfavorable sign. Too large or too small daily variations indicate that the disease process is not terminated. Paracentesis in older children usually causes a drop in temperature; in children aged from 1 to 2 years it scarcely affects the temperature. Antrotomy is necessary in children more frequently than in adults, especially in nurslings. There is usually a lytic drop in temperature after the operation. Complications (sinus thrombosis and meningitis) occur more frequently in children than in adults. In infantile otitis (and sinus thrombosis) there is more frequently an undulating temperature curve without a demonstrable cause. In view of the wide variation in the temperature curve in acute otitis media in children, fever has less value for interpretation of the clinical picture and as an indication for surgical intervention than in adults and may be evaluated only in conjunction with the general and local symptoms.

Editor's Abstract.

Otogenous Abscess of the Temporal Lobe Penetrating the Lateral Ventricle. D. M. Rutenburg, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:97 (June 20) 1933.

Rutenburg states that the extension of an abscess of the temporal lobe into the lateral ventricle may occur without symptoms and does not necessarily constitute a fatal complication. Uffenorde observed two such cases, demonstrated roentgeno-

graphically, only one of which terminated fatally. The author observed the case of a man hospitalized with symptoms of diffuse meningitis who had chronic otitis media and an epitympanic cholesteatoma. Suboccipital puncture yielded a purulent fluid. During a radical operation, a large abscess of the right temporal lobe was drained. At necropsy it was found that the abscess cavity in the temporal lobe communicated with the right lateral ventricle through an opening about ½ inch (1 cm.) in diameter. The ependyma of the right ventricle was covered with fibrinopurulent deposits. The pia mater at the base of the brain was infiltrated with pus. The fact that the extension into the ventricle assumed the form of a fistula and that the edges were callous and surrounded by the same pyogenic membrane that lined the abscess cavity indicates that it had existed for some time; moreover, the fibrinopurulent deposits on the ependyma of the ventricle appeared to be more than several days old. The absence of pus in the other ventricles indicated that the intraventricular foramen had long been closed as a result of the inflammatory process and that the basilar meningitis had spread not along the usual route over Monro's foramen and the third ventricle but probably via the vascular or perivascular route from the choroid plexus. Neither the abscess nor its extension into the ventricle caused symptoms other than headache; the patient had remained at work until the day before hospitalization. The symptoms produced by the spread of a suppurative process into the ventricle may be determined by the nature and rapidity of the extension.

EDITOR'S ABSTRACT.

AN UNUSUAL INJURY TO THE EAR IN MALFORMATION OF THE LABYRINTH. A. RAINER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:112 (June 20) 1933.

Rainer reports the case of a deaf-mute, aged 2½ years, who perforated his left tympanic membrane with a match. Vomiting during the first two days and a flow of clear fluid from the injured ear were the only symptoms until the appearance, on the ninth day, of meningitis which caused death. Microscopic examination of the petrous bones showed luxation of the stapes, with purulent labyrinthitis on the left (injured) side and meningogenic labyrinthitis on the right side. Further, there was a high grade bilateral malformation of the labyrinth of the Mondini type. The malformation of the bony cochlea was characterized by the presence of only two coils, by absence of osseous walls between the individual coils, by imperfect development and partial absence of the septums of the scalae and by defective development and bending of the modiolus. There was a high grade enlargement of the endolymphatic duct and sac. The match had luxated the stapes from the oval window, with rupture of the annular ligament. This permitted the labyrinthine fluid to flow from the ear and also made direct infection of the labyrinth possible. Owing to the malformation of the inner ear, vestibular function was probably lacking; this would explain the absence of the vestibular symptoms usually characterizing labyrinthine injury and the resulting failure to make a diagnosis of labyrinth injury.

EDITOR'S ABSTRACT.

PATHOLOGIC AND CLINICAL ASPECTS OF OTOGENOUS CEREBELLAR ABSCESSES. E. GRABSCHEID, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:160 (June 20) 1933.

Grabscheid reports four cases of otogenous cerebellar abscesses which are atypical in some respects. Two of them originated in acute or subacute otitis, and two, in acute exacerbated cholesteatoma. In one case the first cerebellar symptoms appeared twenty-four days after the onset of the otitis, and the patient died fifteen days later with a fully developed abscess—a rapidity of evolution rarely observed in the cerebellum. The usual intermediate complication of sinus thrombosis did not occur in any of the cases, and otitis interna developed in only one case. In the two cases starting with acute otitis media and mastoiditis, pachymeningitis developed via the subarcuate fossa, spread to the middle and posterior cranial fossae

and led to the formation of a cerebellar abscess. In the other two cases also the development of the abscess must be attributed to pachymeningitis, which originated from the cholesteatoma. In case 1, the abscess was spherical and located above the cerebellar nuclei. In this case there were general symptoms in the form of wasting and polynucleosis, general symptoms of a disturbance in the brain in the form of vomiting and an increase of leukocytes and of albumin in the spinal fluid and local symptoms of involvement of the brain in the form of ataxia, adiadokokinesis, lateropulsion, bradyphasia, past pointing and pressure symptoms. In case 2, the abscess was hook-shaped, the major portion extending between the cortex and the medulla. In this case, the general symptoms of a disturbance in the brain were about the same as in case 1; the local symptoms were ataxia, disturbances in equilibrium, bradyphasia and dysarthria and pressure symptoms. In case 3, the abscess was predominantly cortical; there were severe general symptoms, but no symptoms of involvement of the brain were noted prior to operation except mydriasis on the diseased side. In case 4, the symptoms were still less characteristic before operation. The greater number and the earlier appearance of characteristic symptoms in the first two cases resulted not only from a difference in the involvement of the cerebellar nuclei but from a difference in the speed of evolution of the abscess. When the evolution is slow, as in the last two cases, compensation processes which obscure the symptoms can more readily develop. The fatal terminations must be attributed not only to the surgical failure, but to failure of diagnosis, owing to the unusual pathogenesis without sinus thrombosis or labyrinthitis.

EDITOR'S ABSTRACT.

DISTURBANCES OF COCHLEAR AND VESTIBULAR FUNCTION IN DISEASES OF THE BRAIN. J. KOCH, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:232 (July 14) 1933.

Koch found cochlear disturbances in only twelve of twenty-three patients with diseases of the brain, but all of the patients complained of disturbances in equilibrium. These could be explained and confirmed by spontaneously and experimentally demonstrable deviations of vestibular excitability. Complete loss of vestibular excitability on one side was observed in one case of tumor of the medulla. Normal experimental results in the presence of spontaneous nystagmus were found in two cases of parietal tumor, in one case of cysticercus of the fourth ventricle and in one case of tumor of the vermis cerebelli. In the other seventeen cases there was a difference of vestibular tonus, i. e., a predominance of the nystagmus to one side. Vogel's interpretation of this manifestation as a sign of central vestibular disease is confirmed by the author's observations in cases of tumors of the brain. The disturbance can be produced by the tumor itself, as in tumors of the cerebellopontile angle, the medulla or the cerebellum, or, as is probable in most of the cases, by distant action on the medulla through the pressure-increasing processes in the brain. The frequent occurrence of a difference of vestibular tonus requires the use of rotatory and of caloric tests. Apparently contrasting reactions to the rotatory test and to cold irrigation are explained by the results of warm irrigation. The galvanic test usually merely confirms the results of the other tests. The absence of nystagmus toward the side of the tumor in galvanic tests is a sign of central vestibular injury. The galvanic stimulation is important in the presence of changes in the ear which prevent or influence the caloric tests. The unipolar method has no advantage over the bipolar method. Vogel's statement that in differences of vestibular tonus the latent nystagmus can be activated and made to appear by shaking the head or by the use of weak galvanic currents is confirmed by the author's observations. An excellent testing method is the test for sensitivity to position on Grahe's table, which shows characteristic deviations, especially in tumors of the cerebellum and the cerebellopontile angle. The changes in reaction in cases of tumors of the temporal and parietal lobes and the medulla cannot be definitely interpreted as yet.

EDITOR'S ABSTRACT.

LABYRINTHINE FISTULAR SYMPTOM WITHOUT A FISTULA. A. BLUMENTHAL, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:279 (July) 1933.

Blumenthal reports the case of a man, aged 28, with chronic otitis media and baryecoia of the left side, who sought medical care for a rise in temperature and severe attacks of vertigo with nausea. Examination disclosed absence of the left tympanic membrane and a small bony defect posteriorly above, with an abundant fetid discharge; practical deafness existed on the left side. There was no spontaneous nystagmus. Rotatory and caloric nystagmus were normal. Compression of the air in the external auditory canal produced a distinct nystagmus to the left, starting with a right component, typical of a labyrinthine fistula. Light pressure on the labyrinthine wall produced jerking of the head and severe vertigo. Radical operation disclosed caries of the mastoid antrum. No fistula could be found in the lateral wall of the semicircular canal or in the rest of the lateral labyrinthine wall. The attacks of vertigo disappeared a short time after the operation, but the fistular symptom was still present six and one-half months later. If a macroscopically invisible fistula of the labyrinthine wall had existed at the time of operation this would have been closed with the completion of epidermization, which in this case required nine weeks. The nystagmus produced by increased air pressure in the auditory canal can be accounted for only by softness and consequent flexibility of some portion of the labyrinthine wall which permitted the pressure to be transmitted to the interior of the labyrinth. This softness could be congenital or due to some pathologic process. EDITOR'S ABSTRACT.

Pharynx

UNILATERAL CHOKED DISK DUE TO CHRONIC TONSILLAR INFECTION. G. WILSON and W. F. DARKES, J. A. M. A. **100**:1233 (April 22) 1933.

Two cases are reported in which the author considers optic neuritis to have been due clearly to infection of the tonsils.

The removal of the tonsils, in both cases badly diseased, produced prompt improvement and a return in a relatively short time to normal vision and a normal state of the involved nerve.

If swelling of one or both optic nerves is the only physical sign present, and if the patient does not have headache or other symptoms suggestive of a tumor of the brain, he should not be rushed into submitting to encephalography, ventriculography or a decompression operation.

Cases of the kind reported have probably helped to swell the number of cases referred to naively by the neurosurgeons as instances of "pseudotumor."

ZENTMAYER, Philadelphia. [ARCH. OPHTH.]

EXPERIMENTAL STUDY OF TRAUMATIC TONSILLITIS. C. MIES, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:52 (May 17) 1933.

Mies states that two main theories concerning the etiology of traumatic tonsillitis (tonsillitis after nasal operations) are presented in the literature. One regards traumatic tonsillitis as a secondary infection of the tonsils transmitted from the nose via the lymph channels; the other considers it as a primary surface infection of the tonsils determined by certain factors created by nasal tamponade. In general, a lymphatic connection between the nose and the tonsils is denied since the negative results of Schlemmer's experiments in 1921. Mies conducted experiments on guinea-pigs to determine whether in the pathologic conditions produced by nasal tamponade the flow of lymph was altered. By unilateral nasal tamponade the disturbance of nasal breathing necessary for the occurrence of traumatic

tonsillitis was produced. Following the injections of a dye into the nasal mucosa under these conditions, no dye could be demonstrated in the tonsillar tissue. In simple experimental injections into the mucosa of the nose and palate it was likewise impossible to detect any deposits of dye in the tonsils. This corroborates the results of Schlemmer. In both experimental series, particles of dye were sometimes found in the retrotonsillar tissue on the side into which injection had been made. Animal experiments do not support the assumption of a lymphatic connection between the nose and the tonsils. The Denker theory of the etiology of traumatic tonsillitis, which assumes a secondary infection of the tonsils from the nose via the blood stream, appears to be the most plausible and well founded theory.

EDITOR'S ABSTRACT.

FREQUENCY AND SIGNIFICANCE OF TUBERCULOSIS OF THE TONSILS. THERESE RÖSSNER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:214 (July 14) 1933.

Rössner states that tuberculosis of the tonsils is probably always a secondary infection, originating by the hematogenous or lymphogenous route or in the sputum. The hematogenous route is the most probable in the opinion of the majority of authors. Tonsillar tuberculosis usually assumes a light form, and in the majority of cases it can be recognized only microscopically. Complications following the removal of tuberculous tonsils were rarely seen; in only a few cases was the operation followed by miliary tuberculosis. In the author's clinic, tuberculosis was found in 3 of 296 tonsils examined histologically, or 1.01 per cent. The statistics in the medical literature vary between 5 and 1.6 per cent.

EDITOR'S ABSTRACT.

TONSILLECTOMY OR ELECTROCOAGULATION OF THE TONSILS? HALLE, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:364 (July 22) 1933.

Halle thinks that electrocoagulation of the tonsils, with its unavoidable discomfort for the patient and its injury to adjacent tissue, should be substituted for tonsillectomy only in cases of hemophilia in which the utmost precautions against dangerous hemorrhages are imperative. In all other cases he thinks that electrocoagulation is contraindicated, especially in view of the fact that the dangers of hemorrhage can be almost eliminated by correct surgical procedure. He has designed two forceps which permit a firm grasp of the tonsil even if it is small and embedded. With these the tonsil may be pulled forward so that not much of the glossopalatine arch need be sacrificed in the excision of the tonsil. The technic is briefly described. If one strictly avoids using the instruments for holding the tonsil for handling other tissues or vessels, infection need not be feared, especially if an antiseptic powder is sprayed in the tonsillar fossa after operation.

EDITOR'S ABSTRACT.

QUESTION OF TONSILLOTOMY IN CHILDREN. K. GERLOFF, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:255 (July) 1933.

On the basis of examinations made several years after tonsillotomy in 110 children, Gerloff concludes that the uniform substitution of tonsillectomy for tonsillotomy is not justifiable. Since 76 of the patients became free from symptoms after tonsillotomy, this operation must still be considered preferable to tonsillectomy, a much more extensive intervention and one which deprives the child of a valuable organ. Tonsillotomy is the method of choice in cases involving pure hyperplasia of the tonsils. The anamnesis is of great value in determining whether pure hyperplasia or one of inflammatory origin exists. In the former case tonsillotomy is almost always entirely successful; in the latter case, the result is frequently unsatisfactory. Tonsillotomy should be avoided when the anamnesis and symptoms leave no doubt as to the inflammatory origin of the tonsillar hyperplasia, as examinations

have shown that chronic tonsillitis in early childhood is sometimes unfavorably influenced by tonsillotomy. In such cases climatic or medicinal therapy should be tried, in an attempt to preserve the tonsils, which are necessary for the general immunity of the youthful organism; but in the presence of severe symptoms or at the first appearance of manifestations of central decompensation, tonsillectomy must be employed.

EDITOR'S ABSTRACT.

Larynx

ROENTGEN IRRADIATION IN LARYNGEAL TUBERCULOSIS. BACHMANN and WIEGAND, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:275 (July 8) 1933.

Bachmann and Wiegand report good results with roentgen irradiation of laryngeal tuberculosis. They regard the irradiation as an irritant therapy (inflammation therapy); this conception in general determines the dose. Large doses are to be avoided in tuberculosis. Percutaneous irradiation is the only practical method. Their patients received six doses of 64 roentgens at five day intervals applied alternately to the left and to the right side; the series was repeated once or twice after a six or eight week interval. Most of the patients had severe forms of laryngeal tuberculosis. The first irradiation usually caused the diminution or disappearance of excessive infiltrations of the mucosa, often disclosing previously concealed ulcerations. The hoarseness, dysphagia and other symptoms receded with the decrease of infiltration, and a general and psychic improvement was observed. In no case was an exacerbation of the tuberculosis seen; in the least favorable cases there was merely a standstill. Strongly ulcerating forms were also irradiated with favorable results. The only contraindication to this therapy is a continuous high temperature. The severity of the pulmonary tuberculosis does not determine the prognosis of the laryngeal therapy. Excellent reactions to irradiation of the larynx were observed shortly before death from severe pulmonary tuberculosis. Treatment of the ulcerations with carbon dioxide snow was used with favorable results as an adjunct to the roentgen therapy. Other local therapy may be used. Several pictures showing the progressive results of irradiation are reproduced.

EDITOR'S ABSTRACT.

LATE INJURIES OF THE LARYNX AFTER IRRADIATION. K. VOGEL, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:172 (May) 1933.

Four patients with laryngitis sicca, with superficial ulceration of the vocal cords, had received roentgen or radium irradiation from two to fourteen years previously: in one case for laryngeal carcinoma, in two cases for tuberculous cervical lymphadenitis and in one case for cervical lymphomatosis. Vogel thinks that the irradiation therapy decreased the resistance of the vocal cords to catarrhal infection and produced a tendency to dry laryngitis with torpid superficial ulceration; this is analogous to the acute necrosis of the laryngeal cartilage in irradiated patients. In the patients irradiated in early childhood, hypoplasia of the larynx, in one case producing spontaneous stridor, was noted; also cicatricial hypoplasia of the soft parts of the neck and the inferior maxilla of the irradiated side. Vascular ectasia was seen in the true vocal cords in one case and in the false vocal cords in another case.

EDITOR'S ABSTRACT.

LARYNGEAL TUBERCULOSIS AND LIGHT THERAPY. O. STRANDBERG and J. GRAVESEN, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:245 (July) 1933.

Strandberg and Gravesen state that treatment of laryngeal tuberculosis with light has a much better chance of success if it is accompanied by active pulmonary therapy. In their cases, the light was usually given in the form of carbon arc light baths, and the pulmonary therapy usually consisted of the form of collapse therapy most suited to the case. Of 149 patients who received combined laryngeal and pulmonary treatment in a sanatorium, 80.5 per cent were dismissed with the

laryngeal condition cured. The majority of the cases were severe; in 87 of those in which the tuberculosis was healed, ulcerations had existed. Recurrences were observed in 4 of the 120 cured patients; the others have enjoyed freedom from recurrences for from six months to five years. Fourteen of the patients died of advanced pulmonary tuberculosis without a recurrence of laryngeal symptoms. In 36 patients the pulmonary condition showed a significant improvement and the sputum contained no bacilli; in 57, there was an improvement of the pulmonary and general condition, and in 7, there was an exacerbation of the pulmonary condition. The average number of light baths taken by the cured patients was sixty.

EDITOR'S ABSTRACT.

PHOTOKYMOGRAMS OF VOCAL SOUNDS IN DIPHTHONGIA. W. BERGER, Ztschr. f. Laryng., Rhin., Otol. (Teil 1: Folia oto-laryng.) **24**:264 (July) 1933.

Berger reports his experiences with Frank's photokymographion in the registration of vocal sounds in diphthongia. He thinks that this method of analysis may lay the foundation for a new, exact pathology of the vocal processes.

EDITOR'S ABSTRACT.

Nose

OSSIFICATION OF THE FALX CEREBRI AND SINUSITIS. G. WORMS, Rev. d'oto-neuro-opht. **10**:729 (Dec.) 1932.

In the great majority of instances ossification of the falx cerebri evolves without symptoms, although exceptionally it may by its size produce signs of intracranial hypertension, betrayed by deep, dull headaches. The author's observations comprise the cases of six patients in whom the ossification was revealed by roentgenograms during hospitalization on account of other conditions, or who complained of symptoms that could be attributed to lesions of the dura but were in reality due to paracranial infectious foci. In one case the most painstaking and repeated examinations were necessary to discover an infection of the frontal sinus and to avoid an intracranial operation. The cause of the meningeal alterations is difficult to determine, but the coincidence of infected nasal sinuses is significant. Since symptoms from the ossification alone are so rare, the relation of cause and effect between the dural anomaly and the headache must be mistrusted. A concomitant involvement of the sinus must be carefully sought.

DENNIS, Colorado Springs, Colo. [ARCH. NEUROL. & PSYCHIAT.]

ACUTE PURULENT FRONTAL SINUSITIS WITH OCULO-ORBITAL COMPLICATION: CEREBELLAR AND LABYRINTHINE PHENOMENA; OPENING OF BOTH FRONTAL SINUSES AND EXPLORATION OF THE RIGHT FRONTAL LOBE OF THE BRAIN; FIXATION ABSCESS; CURE. CANUYT, LACROIX and SCHEPENS, Rev. d'oto-neuro-opht. **10**:735 (Dec.) 1932.

A young girl, with an acute exacerbation of chronic frontal sinusitis on the right side but with no cerebellar or labyrinthine signs, was operated on, and external drainage was instituted. One week later heaviness of the head, vertigo and vomiting appeared, and the patient was unable to stand. Lumbar puncture relieved the symptoms temporarily and revealed a normal fluid, except that the pressure was slightly increased. Neurologic examination revealed prostration, a tendency to fall backward, an incorrect result in the heel-to-knee test, a bilateral Kernig sign, slight stiffness of the neck, vertigo, nystagmus to the left in all positions of the eyes, incontinence and difficulties of speech. Nineteen days after the original operation the left frontal sinus was opened, and the right frontal lobe was explored, with negative findings. A fixation abscess (aseptic abscess) was

produced and opened five days later. Immediate improvement began, and recovery was rapid. There is no consensus as to the operative treatment in cases of acute suppuration of the frontal sinus with orbital complications. The authors believe that the sinus should be opened promptly and widely, with external drainage and without curetting the sinus walls or the nasofrontal duct. The curative action of fixation abscess in grave septic conditions is extolled.

DENNIS, Colorado Springs, Colo. [ARCH. NEUROL. & PSYCHIAT.]

RETROBULBAR NEURITIS AND DIPLOPIA, WITH SIGNS OF FRONTAL SINUSITIS: OPERATION; ABSENCE OF SINUSITIS; RECOVERY. J. SOUCHET and A. LACROIX, Rev. d'oto-neuro-opht. **11**:26 (Jan.) 1933.

This observation concerned a woman, aged 52, who, following a slight coryza, suffered violent pain in the left supra-orbital region, accompanied by exquisite tenderness over the outer part of the frontal sinus area in the forehead and in the orbit. A roentgenogram revealed cloudiness over the left frontal sinus, more marked over the lateral part and extending beyond its boundary, and cloudiness of the left maxillary sinus. Examination of the eyes showed diplopia, limitation of movements, a small central scotoma for colors, a contracted visual field and a visual acuity of 1/10 in the left eye. Opening of the sinus revealed an apparently normal cavity. Relief from pain was prompt, and vision rapidly became normal, although a transitory spasmodic myopia occurred in both eyes during convalescence. Punctures of the left maxillary sinus gave negative results. The tenderness over the forehead is explained by periostitis, and it is assumed that the origin of the retrobulbar neuritis was the coryza, the infection passing over the sinus itself and attacking the surrounding periosteum and dura. From this point it could readily reach the optic nerve by means of the cellular tissue. In 1921, Lemaitre thought that many cases of retrobulbar neuritis arose in this manner. The entire question is still too vague to permit of more than an expression of opinion.

DENNIS, Colorado Springs, Colo. [ARCH. NEUROL. & PSYCHIAT.]

PATHOGENESIS AND THERAPY OF ALLERGIC INFLAMMATION OF THE ACCESSORY NASAL SINUSES. A. GLASSCHEIB, Monatschr. f. Ohrenh. **67**:565 (May) 1933.

Glasscheib says that the allergic catarrhal inflammation of the nose, which is characterized roentgenologically by edematous swelling of the mucosa of the maxillary sinus to the point of polypus formation, has its origin in a purulent inflammation of the maxillary sinus. Some of the pus secreted by the mucosa is resorbed, especially when there is stasis, and with it the toxins and products of protein decomposition from the disintegrated bacteria. These constitute antibodies; the union of antigen and antibody leads to the development of allergy within about eight days. The reaction of the secretion is alkaline instead of acid as in most allergic secretions, but the eosinophilic reaction of the tissue of the mucosa and the polypi as well as that of the blood is a certain sign of allergy. This theory of pathogenesis has a therapeutic interest. In the treatment of acute suppuration of the maxillary sinus, resorption of the pus should be prevented by aspiration or irrigation. If an allergic inflammation does develop, desensitization with albumin should be instituted. The administration of acid may also be tried. If the polypi do not regress with this therapy, curettage may be tried, followed by desensitization. The inflammation usually extends to the ethmoid, frontal and sphenoid sinuses, but is most easily recognized in the maxillary sinuses. The disease passes imperceptibly from the acute to the chronic stage, and its early recognition is therefore all the more important.

EDITOR'S ABSTRACT.

ASPIRATION OF ALAE NASI. AURÉL RÉTHI, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:183 (June 8) 1933.

Aspiration of the alae nasi during normal respiration so that they lie against the septum constricting or closing the nares may be due to weakness of the cartilaginous framework of the alae nasi or to a stenosis at the anterior nares owing to thickening of the membranous septum. In the latter case Réthi employs Halle's method of removing the thickened or deflected cartilage and the connective or lipoid tissue by an incision into the membranous septum behind the nares. If the condition is caused by the looseness and weakness of the cartilaginous framework of the alae nasi itself, he uses one of two methods. If the aspiration affects the alae nasi as a whole, he removes a fusiform section of cartilage and skin from the interior surface of these structures by parallel incisions made obliquely upward. This is done without injury to the exterior wall. After suturing of the wound the inner wall of the alae nasi is taut, and there is a gap between it and the outer skin surface. The taut inner wall, its rigidity being increased by the scar tissue, prevents the aspiration of the alae nasi. The author used this method successfully in five cases. In cases in which it is chiefly the free edge of the alae nasi which is drawn in, he makes an incison in the nasal fold, which stops about 3 mm. from the free edge of the alae nasi. By means of another incision a crescent-shaped piece of skin is removed without injury to the interior wall. When the suture is applied the anterior portion of the alae nasi is drawn well away from the septum. Three patients were treated in this manner, with good therapeutic and cosmetic results.

EDITOR'S ABSTRACT.

Miscellaneous

THE FOREIGN BODY IN THE FOOD OR AIR PASSAGE. D. A. CROW, Practitioner **130**:369 (March) 1933.

Crow strongly urges that a foreign body in the esophagus which does not naturally pass down in six hours be removed by an experienced esophagoscopist. The degree of urgency depends somewhat on the sharpness of the swallowed object, since the wall of the esophagus is so thin that a pin or a bone may work its way through in a short time and may be detected only after a mediastinal abscess has formed. In the case of a foreign body in the air passage (except when complete laryngeal obstruction has occurred) the procedure is as follows: (*a*) the taking of a careful history, with special reference to the cough at the moment of inspiration; (*b*) an indirect laryngeal examination; (*c*) a thorough physical examination of the chest; (*d*) examination with the x-rays, and (*e*) bronchoscopy. Except in cases of almost complete obstruction, immediate bronchoscopy is not essential, as the trachea and bronchi are cartilaginous tubes and are not liable to instant perforation. Instructions for incising the cricothyroid membrane to relieve the complete blockage of air are given in detail.

The author believes that a foreign body in a bronchial tube will seldom be coughed up; hence more than a short delay before examination by bronchoscopy is not justified.

In regard to prophylaxis, the following suggestions are made: 1. Pins, nails, coins and other articles should not be held in the mouth. 2. Dentures should fit properly. 3. Whooping cough and postanesthetic vomiting should be carefully treated. 4. The posterior part of the pharynx should be protected when an anesthetic is given for dental extraction.

SAPPINGTON, San Francisco. [AM. J. DIS. CHILD.]

LEUKEMIC CHANGES IN THE UPPER RESPIRATORY TRACT. H. GLÜCKERT, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:135 (June 20) 1933.

Glückert says that leukemic changes in the upper respiratory tract have received little attention because they are comparatively rare and also because the symptoms

in the skin and in the internal organs usually dominate the clinical picture. He describes in detail two cases of acute and two of chronic lymphatic leukemia with involvement of the upper respiratory passages. In the two acute cases the hemorrhagic diathesis which is exhibited chiefly in the lips, gums and oral mucosa was the main symptom. In the first case the entire upper lip, the nares and the right side of the palate were involved. In the second case swelling of the lymph nodes and facial paralysis on the left side appeared first; later hemorrhages from the mouth, nose and upper lip appeared, as well as a tumor-like swelling of the nasopharynx which was diagnosed at biopsy as a lymphocytic infiltration. One of the chronic cases was characterized by dyspnea due to a laryngeal tumor. The condition was first diagnosed as a round cell sarcoma but at biopsy following a second tracheotomy it was demonstrated to be a lymphocytic infiltration. In the fourth case lymphocytic infiltrations of the skin affecting chiefly the nose, cheeks, chin and ears were the main symptom. In all chronic inflammatory processes of the upper respiratory tract, the blood picture should be studied, as it usually permits the diagnosis of leukemia. If tumor-like swellings are present, early biopsy is advised, as typical histologic changes appear in leukemia. Subacute inflammatory symptoms in the pharynx are secondary manifestations of the lymphatic leukemia; the pharynx is not to be regarded as the primary seat of the disease. Roentgen irradiation of the long bones is not effective in acute leukemia. In chronic leukemia the result of irradiation appears to be better. Local treatment in the upper respiratory passages is unnecessary, with the exception of tracheotomy in case of laryngeal stenosis resulting from the formation of a tumor.

EDITOR'S ABSTRACT.

ENDOCRINOLOGIC DIAGNOSIS IN OTOLARYNGOLOGY. M. BREITMANN, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:289 (July 22) 1933.

Breitmann thinks that a systematic endocrinologic examination in otolaryngologic diseases may lead to good diagnostic and therapeutic results in cases in which the customary methods of examination and treatment fail. It is not sufficient to find out which glands exhibit dysfunction. It is necessary to consider the interrelation of the glands and to determine which of the endocrine dysfunctions in the whole chain of reciprocal relations of the endocrine glands is the primary one. Only the treatment of the primary endocrine disease can produce successful therapeutic results.

EDITOR'S ABSTRACT.

Society Transactions

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OTOLARYNGOLOGY

Feb. 15, 1933

B. Parrish, M.D., *Chairman*

Otitis Media in Scarlet Fever. Dr. H. J. Williams.

The object of this report is to discuss the aural complications occurring in patients with scarlet fever during the years from 1922 to 1929 at the Philadelphia Hospital for Contagious Diseases. It seems essential to understand that the Philadelphia Hospital for Contagious Diseases is municipally owned, so that the overwhelming majority of the patients admitted were from the less well-to-do and the laboring classes. The patients, therefore, on admission often suffered from inadequate medical and hygienic care and from improper, if not insufficient, nourishment.

During these eight years, a total of 14,733 patients with scarlet fever were admitted to the hospital. In 1,535, or 10.8 per cent, suppurative otitis media developed. One third had bilateral involvement. This compares favorably with figures from other sources. The mean average given by other investigators is about 15 per cent. In 167, or 10.8 per cent, of the 1,535 patients with otitis media, mastoiditis that necessitated simple mastoidectomy developed. The incidence of mastoiditis in the total number of patients with scarlet fever was 1.1 per cent. In 157 of the 167 patients with mastoiditis, the condition was unilateral, and in 10 it was bilateral. There were 18 deaths among the patients operated on for mastoiditis—a percentage of 10.8. The mortality for the 1,535 patients with otitis media was 4 per cent. The average number of days from operation to recovery was forty-seven and eight tenths in 148 cases in which the time of recovery was recorded.

The incidence of surgical mastoiditis in three different groups of patients did not vary widely: It was 6.6 per cent in those in whom the tympanic membrane was incised before rupture, 7.7 per cent in those in whom the membrane was incised after rupture and 9.3 per cent in those in whom the membrane ruptured and was not incised. From these figures the conclusion may be drawn that early incision of a bulging tympanic membrane before it ruptures and incision of a ruptured membrane when the rupture is inadequate for proper drainage tend to lower the incidence of acute surgical mastoiditis. Repeated incision of the tympanic membrane is seldom of value, provided that the original incision is free. Incision of a tympanic membrane which has already ruptured, but in which the opening is inadequate for proper drainage, is of distinct value and a good practice.

Concerning the incidence of otitis media according to age groups, 61.7 per cent of the patients were 5 years old or younger; 31.2 per cent, between 5 and 10 years; 4.2 per cent, between 10 and 15 years; 2.1 per cent, between 15 and 20, and 1.8 per cent, over 20. According to statistics generally, the largest percentage of cases occurs in children under 10 years of age.

The anatomy of the eustachian tube in young persons favors the development of acute otitis media and its continuance into the chronic form. At birth, the eustachian tube measures not more than 20 mm. in length and is practically straight. In the adult, it measures about 36 mm., and its course is moderately tortuous. In children, the pharyngeal orifice and the caliber of the tube are as large as in the adult, and the pharyngeal orifice is nearer the floor of the nose

than in the adult. These factors are supplemented by the occurrence of faucial and pharyngeal tonsils. Of the 1,535 patients with otitis media under discussion, only 55, or 3.5 per cent, had these structures removed prior to the development of scarlet fever. Of the total number of 14,733 patients with scarlet fever, the pharyngeal and faucial tonsils were absent in 861, or 6.8 per cent. The percentage is about twice as large in the patients who did not have aural complications as in those who did.

Contrary to the opinion of Layton, Dr. Williams believes that sinusitis in scarlet fever is common and predisposes to acute and chronic otitis media. There is a marked seasonal variation in the occurrence of scarlet fever and, therefore, of scarlatinal otitis. The incidence is greatest usually from the middle of January to the middle of May. But the yearly variation in otitis media is more marked than that in the total number of patients with scarlet fever. It is Dr. Williams' opinion that the concurrence of another exanthem with scarlet fever increases the incidence of aural complications, and that the increase is more marked with measles than with other exanthems.

Dr. Williams is in complete accord with the assertion of Borden that "it may truly be said of scarlet fever that aural complications of any degree of severity may arise at any time from the first day of the acute symptoms to the last day of convalescence." He also agrees with Borden and Layton that there is no great tendency of the disease to extend from the bone to the meninges, as shown by the occurrence of only 7 cases of meningitis among 14,733 patients with scarlet fever. In all of the patients with meningitis except 1, the faucial and pharyngeal tonsils were present.

Patients in whom mastoiditis develops early are likely to show fever, localized pain and tenderness, some sagging of the posterosuperior wall of the canal and moderate or excessive discharge. Those in whom it develops late are likely to present marked edema of the tympanic membrane, sometimes with a nipple-like perforation, sagging of the posterosuperior wall of the canal, profuse and persistent discharge, usually without pain or fever, and frequently moderate postauricular edema and redness progressing rapidly to a subperiosteal abscess. Discussion of roentgenographic and laboratory diagnosis is purposely omitted. The useful principles of treatment which are available today are essentially the same as those available twenty years ago.

SINUSITIS IN INFANCY AND EARLY CHILDHOOD. DR. WALTER L. CARISS.

Opinions as to the frequency of occurrence, methods of diagnosis, prevention and treatment of sinusitis in infancy and early childhood are at wide variance. Davis and Dean recently reported that the condition occurs frequently. Campbell said: "The child who has reached the age of 1 year without sinusitis does not live." Two leading pediatricians, on the other hand, said: "Sinusitis of infancy is of rare occurrence."

The great discrepancy in reports as to the incidence of sinusitis at this age is doubtless due to difference of opinion as to what constitutes sinusitis. More exact methods of diagnosis and more general realization that infants do have sinusitis are responsible for the greater number of cases reported; for when sinuses are present, sinusitis is always possible. In infancy ethmoid involvement is the most common type, and maxillary involvement is next in frequency. Infection of the frontal and sphenoid sinuses is of such infrequent occurrence as to be practically negligible.

The aids in diagnosis of the disease in young children are limited. The history and symptomatology are uncertain. Direct inspection is difficult and frequently of little help on account of the small nares. Bacteriologic and otologic studies are difficult for the same reason, although Dean reported that they are of much aid. Even roentgenograms are hard to obtain and should always be properly correlated with the other findings. Transillumination is of much less value in infants than

in adults. Nasopharyngoscopic examination, on the other hand, is usually readily made, even in very young infants, and is of much assistance.

Complications are frequently present, the most frequent being otitis media, tonsillitis, retropharyngeal and peritonsillar abscess, laryngitis, bronchitis, pneumonia, peribronchial lymphadenitis, cervical adenitis, malnutrition, arthritis, pyelitis and anemia. Orbital cellulitis or orbital abscess is not an uncommon complication, as reported by Porter, and as shown by several of Dr. Cariss' cases.

Treatment must vary with the individual patient. Undoubtedly a diet rich in vitamin A is of value in stimulating the resistance of mucous membrane of the respiratory tract to infection and in aiding recovery after infection has occurred. The work and reports of Dean, DeSanctis, Shurly and Carmack have called attention to this. Cody reported that avitaminosis presents essentially the same nasal symptoms as infection, with which it may readily be confused.

In regard to local treatment, there is unanimity of opinion as to the necessity for aeration and drainage, but the value of the local use of drugs, oils, ointments, bacteriophages and other remedies is much in dispute. The reports of Hilding, Proetz and others on the ciliary action of the nasal and sinus mucosa bring one to the realization that measures to preserve and restore this action are the most effective means of treatment. In the hyperplastic cases, mild astringents and mild suction, in addition to gentle douching or swabbing, are necessary. Contrary to some opinions, Dr. Cariss found that ultraviolet irradiation was of benefit. Vaccines, though occasionally of benefit, are usually disappointing. Allergic and endocrine factors must be treated as such.

In the acute and subacute cases without complications surgical intervention should be withheld. With persistence of symptoms or development of complications, operation is indicated. This should be as conservative as is possible with thoroughness.

In the case of the antrum, infraturbinal puncture with washing will usually suffice. Gentle intranasal curettage of the ethmoid labyrinth is frequently necessary. An attempt should be made to preserve the turbinates, and it is important to cause as little destruction of the membrane as possible.

When local complications develop, such as orbital cellulitis or orbital abscess, more radical and prompt operation is essential, and external drainage of the antrum or of the ethmoid sinus is often necessary. The type of treatment or operation indicated in such cases must depend on the conditions present, as shown in the following cases of orbital involvement.

CASE 1.—M. W., aged 4 years, previously well, complained of severe frontal headache on the left side three days before admission to the hospital. The next day, the mother noticed swelling about the left eye. On admission, the temperature was 102.4 F. The upper and lower lids of the left eye were red and edematous. There was a slight mucoid discharge from the left nostril. The left middle turbinate was swollen and inflamed. Roentgen examination showed hazy left ethmoid sinuses. Internal strabismus developed on the day following admission. Otherwise ocular examination gave negative results. Local treatment consisted of applications of a weak solution of epinephrine hydrochloride and the use of a mild astringent oil with an ice bag externally. The patient was discharged in five days with the eye normal and the ethmoiditis improved.

CASE 2.—A. E., a rachitic colored child, aged 2 years, was admitted to the hospital with a diagnosis of gastro-intestinal toxemia and bronchopneumonia. No particular symptoms developed in the nose or sinuses until fifteen days after admission, when the nostrils were found to be filled with a purulent discharge; the mucous membrane was inflamed; the tonsils were diseased, and some cervical adenitis and inflammation of the left tympanic membrane were noted. There were no ocular symptoms. Two days later, there were gross edema of the upper and lower lids of the right eye and marked exophthalmos. Intranasal curettage of the right ethmoid sinus was done, and the left ear drum was incised. There was almost immediate improvement in the ocular condition, and within ten days the eye was normal.

CASE 3.—R. L., aged 6 weeks, presented a particularly interesting case because of his age and the extent and the persistence of the orbital infection, with eventual recovery. The child was well until two days before admission, at which time acute rhinitis developed. Two days later, both lids of the left eye became inflamed and edematous and there was some exophthalmos. On the advice of Dr. Langdon, the left upper lid was incised, with release of some purulent discharge. On the following day, the orbital inflammation had increased and there was tremendous exophthalmos with marked ectropion of the upper lid. The nose showed marked swelling of both lower and middle turbinates with a mucoid discharge in the region of the left middle turbinate. The child's condition was such that immediate drainage was indicated. External opening and drainage of the ethmoid sinuses were done, and considerable pus was present. An incision was also made in the left upper eyelid at the orbital margin, and a drain was inserted. Hot compresses were applied externally and inhalant drops of ephedrine were used in the nostrils.

The condition improved locally and generally so that the ectropion and exophthalmos practically disappeared in ten days following operation. For four months, there were exacerbations of the orbital condition with recurrent exophthalmos. Cellulitis of the lids developed at times, together with ectropion of the conjunctiva and muscular imbalance, necessitating numerous incisions. The zygomatic area of the infra-orbital wall was curetted several times, and a sequestrum of bone was removed. Intranasal curettage of the ethmoid region was performed. A sequestrum of bone was removed through the external ethmoid incision many weeks after operation. During the acute exacerbations, cold applications did not help the local condition, which, however, seemed to improve with hot compresses. The infant was discharged four months after admission with normal vision and ocular movements, a normal nasal condition and a steady gain in weight.

The maintenance of a nutritional maximum and the stimulation and preservation of normal ciliary action of the nasal and the sinus mucous membrane constitute the best line of defense and the strongest base of attack in the treatment of sinusitis in infants and young children. When these defenses are destroyed, surgical treatment is indicated, which should be as conservative as is possible with thoroughness.

SPONTANEOUS CEREBROSPINAL OTORRHEA. DR. ORAM R. KLINE, Camden, N. J.

It has long been known that cerebrospinal fluid may escape from the ear after fractures of the base of the skull or following operations during which the inner ear or the subdural space has been opened, but the literature dealing with spontaneous cerebrospinal otorrhea is apparently meager.

Canfield, in 1913, reported a case of discharge of cerebrospinal fluid and pus from the ear with no history of trauma. The autopsy report in this case described a perforation through the wall of a cell at the apex of the right temporal bone which permitted the entrance of infection to the subdural space and the escape of cerebrospinal fluid into the pneumatic structure of the temporal bone and thence to the middle ear and the external canal. In the same paper, Canfield reported certain findings during an operation on the labyrinth which are of interest in connection with the case presented here. Cerebrospinal fluid escaped from a small circular opening in the dura, which was probably a large perilymphatic duct opening atypically into the subdural space. Although the hole in the dura was obliterated by a suture, the postoperative flow of cerebrospinal fluid was profuse and persistent until a graft from a dog's dura was tamponed over the opening. After this procedure the patient made a complete recovery.

Roth, in 1930, reported the case of a patient, aged 53, who had complained of a discharge from the left ear since childhood. About six years before examination there had been two operations on the left mastoid. Attacks of vertigo persisted until after the second operation. The discharge was not profuse. The left labyrinth was less sensitive to stimuli than the right, and the response to the fistula test was not constant. The history suggested that the labyrinth was

wounded during the mastoid operation, and this case should be classified as one of postoperative cerebrospinal otorrhea.

Although the literature has been carefully searched, these were the only records found of cases which seemed to resemble the one reported here.

K. W., a man, aged 54, was examined on Dec. 30, 1932, because of a watery discharge from the right ear. About three hours before, he stooped to pick up an object from the floor and suddenly felt warm fluid run from his right ear. Careful questioning failed to bring out any other symptom of illness. He did not recall ever having had an earache or a discharge from the ear, although he had always been slightly deaf on the right side. Later, a history of an abscess in the right ear complicated by erysipelas at 9 months of age was obtained; the discharge did not persist for a prolonged period. He had always been healthy except for typhoid and rheumatism at the age of 30 and an occasional "bilious attack." On examination, the right external auditory canal presented a clear, watery discharge which was so profuse that it was dropping from the lobe of the ear. Coughing caused a definite increase in the flow. About 3 cc. of the fluid was collected for examination and was reported to be clear and to respond to all tests in a manner characteristic of cerebrospinal fluid.

There was no gross loss of hearing. Two exostoses of the inferior bony wall of the canal reduced the natural lumen approximately 50 per cent. Beyond the exostoses, a gray area could be seen which resembled the drum membrane, although no landmarks were visible. The point of entrance of the fluid into the canal was not identified at this time. In three or four days the discharge ceased, and the patient said that he had never felt better. About 2 p. m. the next day, he became nervous and restless. Soon afterward he complained of headache, and in three hours he had a temperature of 103 F. and severe frontal headache. The membrane visible beyond the exostoses was gray and apparently presented no signs of inflammation. That evening, he was completely irrational. The neck was stiff, and the Kernig sign was suggestive. The drum membrane at this time presented a blister in the region of the rivinian segment, but the canal was still dry. When the blister was broken by wiping with a cotton applicator, clear fluid began to flow and continued until death. During the night, a twitch of the right side of the face developed, which was evidently due to irritation of the facial nerve. Culture of the fluid showed Micrococcus flavus. The white blood cells numbered 24,050 with 91 per cent polymorphonuclears.

At autopsy, there was a dense suppurative exudate over the vertex and the frontal lobes, with some involvement of the vermis. Both temporosphenoid lobes were grossly free from exudate. No tumors or abscesses were found in the brain, and the pathologic changes were apparently confined to the meninges. A cross-section of the cord at the foramen magnum appeared normal. On the superior surface of the right temporal bone, between the arcuate eminence and the petrosquamous suture, there was a circular opening in the dura and bone about 2.5 mm. in diameter. The dura surrounding this opening was smooth and free from adhesions, and seemed to dip down into the opening. A probe placed in the opening passed through a small fistulous tract into a sac attached to the posterior bony wall of the tympanum. The walls of the sac and of the tract consisted of soft, friable, brownish tissue. No pus or gross signs of inflammation were found in the mastoid. Pus taken from the exudate over the vertex yielded a pure culture of pneumococci. A section from the fistula showed brain tissue, in the center of which appeared a canal about 0.25 mm. in diameter. This fistula may possibly have been the duct of a congenital cyst. There was no evidence of inflammatory tissue around the cyst.

After going over the data in this case, Dr. Eagleton expressed the opinion that infection of the sphenoid sinus should be considered as the possible focus of infection resulting in meningitis, and that the rupture of the membrane permitting the escape of cerebrospinal fluid into the external auditory canal was possibly incidental. Unfortunately, the sphenoid sinus was not opened at autopsy, so this theory cannot be confirmed. Let it be assumed, however, that a pneumococcic

embolus from the sphenoid sinus entered a pial vessel, causing a local abscess within the arachnoid. This irritation of the meninges resulted in an increase in volume of the cerebrospinal fluid, but before the intracranial pressure had become sufficient to cause definite headache the degenerated walls of the congenital cyst gave way, permitting the excess fluid to escape by way of the external auditory canal. On the other hand, if the degenerated sac ruptured spontaneously, it is not altogether illogical to assume that pneumococci were introduced into the aural canal by a handkerchief contaminated with nasal secretions and thence entered the subdural space, causing generalized meningitis immediately. Since bacteria entered the subdural space directly rather than from an overlying infection, a local reaction at the place of entrance would not necessarily occur.

DERMOID CYST OF THE NASAL DORSUM. DR. ROMEO A. LUONGO.

This paper was published in full in the ARCHIVES (**17**:755 [June] 1933).

DISCUSSION

DR. C. V. BATSON: I was impressed by the interorbital width in this case, and by the fact that all the requirements for a dermoid cyst were fulfilled. The hair found in the cyst was similar to that of the lip rather than that of the nose. In fetal life there is a median groove, and the single frontal process must be thought of as in two halves which become fused.

March 15, 1933

B. PARRISH, M.D., *Chairman*

AIMS AND ACTIVITIES OF THE JOHNS HOPKINS OTOLOGICAL RESEARCH LABORATORY. DR. SAMUEL J. CROWE, Baltimore.

The work in this laboratory is divided into several parts, but the principal aim is the correlation of normal and pathologic hearing. It is important to limit the studies to only a few deaf people. Since in the Johns Hopkins Hospital and the Baltimore City Hospitals there are about 1,200 autopsies a year, every one is tested who might come to autopsy. To accomplish this, cooperation is necessary between the various staffs and, when the condition of the patient permits, hearing tests are made in a sound-proof room.

Since 1924, 10,000 audiometer records have been made, and 1,500 temporal bones have been sectioned serially. Records of hearing and acuity have been made for 7,000 patients. Correlations with pathologic conditions were made: (1) to determine the loss of perception of high tones (Dr. Polvogt will explain what may be expected from such study) and (2) in connection with lesions of the middle ear. The work of Weaver and Bray was a godsend. A large number of temporal bones associated with various degrees of impaired hearing have been accumulated.

Owing to the development of the audiometer, my associates and I have been able to make quantitative estimations and to test high as well as low frequencies. With the tuning fork, the high limit is one third off, and with the monochord and the Galton whistle, one cannot determine the degree of impairment.

In recent years we were fortunate in securing the services of Dr. Guild of the University of Michigan. Even after he came it required four years to develop a method good enough to section the acoustic hair cells. After that we began to make correlations and to find some of the most common types of deafness.

THE ANATOMIC BASIS FOR THE IMPAIRMENT OF LOSS OF PERCEPTION OF HIGH TONES. DR. LEROY M. POLVOGT, Baltimore.

For three years my associates and I have been correlating the hearing tests of patients with impairment or loss of high tones and the histologic condition of their middle and inner ears. During this period we have examined serial sections of the temporal bones of several hundred patients. In this group we have been able to find only 60 patients suitable for such a study, who were tested under more or less ideal conditions. These conditions were: First, the general health of the patient was such that he was alert and willing to cooperate with the tester; second, the test was made in a sound-proof room or a reasonably quiet place; third, the time that elapsed between the tests and the securing of the temporal bone was within from a few days to a few months; fourth, the temporal bone was removed and put in a suitable fixing fluid shortly after death, and fifth, the fixation and the histologic sections were good enough to enable one to form some opinion as to the condition of the finer nerve fibers and end-organ structures. For various technical reasons, only 99 ears were available for study. In 64 of these, the middle ear was normal; in 35 there was evidence of an old otitis media, which, however, did not seem to have any direct bearing on the impairment or loss of perception of high tones. In addition to the hearing tests, a complete history was taken and general physical examination, clinical laboratory tests and an autopsy record were made in every case. It is apparent that the loss of high tones in the cases reported was not due to a general systemic disorder, such as syphilis, arteriosclerosis or nephritis. In fact, we have no idea of the etiologic factor, unless it was a focal infection, such as abscesses of the teeth or chronic infection of the upper respiratory tract.

In testing the hearing of these patients, not only is it necessary to make a good tuning fork test, but additional tests must be made with the audiometer and the monochord. If the examination is limited to the use of tuning forks, no knowledge is gained of frequencies above 4,096, and therefore no information is secured regarding the condition of the nerve or the end-organ in one third of the entire length of the cochlea. The advantage of using an audiometer is that the degree of impairment is accurately determined, and it is by this accuracy of measurement that the correlation between function and lesion is made possible.

Early in our study, it became evident that the important pathologic process affecting perception of high tones is located in the basal coil of the cochlea, and chiefly in the lower half of the basal coil. A detailed graphic reconstruction of all abnormalities seen in the study of serial sections of the cochlea was made for each of the 99 ears. In most cases in which there is impairment or loss of high tones, one finds damage to the end-organ or to the nerve. The end-organ, or the organ of Corti, is attached to the basilar membrane and is in the cochlear duct. This end-organ, therefore, is surrounded by endolymph, which is secreted by the stria vascularis and, as far as present knowledge goes, is its sole source of nutrition. When there is extreme atrophy of the stria vascularis, there must be some change in the endolymph and in the nutrition of the organ of Corti, which decreases the normal irritability of the hair cells and thus decreases the acuity of perception of the high tones normally heard in the affected area. The anatomic changes found in the organ of Corti vary in degree from atrophy of the hair cells alone to atrophy of the entire end-organ. There are also varying degrees of atrophy of the nerve in the bony spiral lamina.

From microscopic sections one may gain an anatomic basis for the loss of high tones, namely, damage to the nerve or a combination of damage to the end-organ and to the nerve. When one groups a number of cases in which the power of perception of tones above a certain octave has been lost, this group should show a similar pathologic process. If this is true, one is justified in thinking of certain areas of the cochlea as associated with perception for definite tones.

EXPERIMENTAL STUDIES OF THE PHYSIOLOGY OF THE EAR. DR. WALTER HUGHSON, Baltimore.

During the past two years, investigations have been carried out on the cat's ear to determine the normal physiology and function of its individual parts. This study has been made possible by the use of the phenomenon of change in the electrical potential set up in the auditory nerve by stimulation of the ear with different types of sound. In all experiments this stimulation has been produced either by an oscillator or by a heterodyne audiometer, both of these methods producing pure tones of different frequencies, and also by a phonograph record. The electrodes are placed on both auditory nerves by introducing them through small trephine openings over the lateral lobes of the cerebellum. The ground electrode is placed in the muscles of the neck. By the mere turning of a switch the lead can be brought off from either the right or the left nerve. The electrodes are connected with a five stage amplifier in a room 60 feet (18 meters) from the operating room. Here also is a comparison amplifier set which produces a constant intensity of sound. Between the sets is placed an attenuation apparatus which reduces the intensity of tone passing through the comparison amplifier in decibels of attenuation. The observer, by listening first to the cat system and then to the comparison system, can reduce the latter so that the intensity of tone coming through the two sets is brought to a balance. In all experiments the left ear is used as the normal control.

The effect produced by immobilizing the membrane of the round window in either an acute or a chronic experiment has been reported. During a year an effort was made to determine what particular type of experimental lesion reducing the intensity of tone transmitted might be benefited by immobilization of the membrane of the round window. The surprisingly slight degree of deafness produced by an infection of the middle ear in certain cases is a common observation in clinical otology. When sections of these ears are examined, it is found that though many adhesions may be present in the attic there is also a mass of adhesions in the round window niche. On the other hand, when marked deafness follows infection, sections demonstrate that the pathologic changes are confined to the structures in the attic and that the round window niche is unaffected. An experiment was therefore carried out in which an ointment having about the consistency of pus was injected into the middle ear. All tones were markedly affected, the degree depending on the pressure with which the ointment was injected. In the cat the round window is separated from the middle ear by a bony septum. When a pledget of cotton was placed in the round window niche either before or after the injection of the ointment, the reduction in intensity was far less than that found when the round window niche was left open.

In another series of experiments an effort was made to determine the function of the stapedius tendon. This was divided in acute experiments, and animals were also operated on under aseptic conditions and kept for varying periods. No significant effect could be demonstrated as a result of the division of the tendon in either of the two groups. In the acute experiments a tiny hook was placed about the tendon and weights were applied to a thread leading over a pulley, thus increasing the tension of the tendon. Depending on the amount of weight used, the higher and lower tones were either reduced or lost; only the middle range remained, and these tones also were considerably below normal. When the weight was removed, the intensity of tone immediately returned to its original level. As in similar experiments on the tensor tympani muscle, no effect could be observed following the experimental procedures except the effect produced by extreme tension. It would seem, therefore, that these muscles function only in some protective manner when they are called on to immobilize the ossicular chain almost completely. This reflex might function, for instance, in the presence of loud noises.

Another investigation has been carried out which has reached a point warranting a preliminary report. Many experiments have been performed in an effort to localize the areas in the cochlea at which certain tones are heard. These experiments were made either on animals with conditioned reflexes or by using

the aural reflex of the guinea-pig. Furthermore, in all of these experiments the cochlear wall was actually opened and escape of fluid occurred. In experiments reported a year ago it was definitely demonstrated that when fluid was permitted to escape from the cochlea there was marked and rapid reduction of intensity of tone, depending on the amount of fluid which had escaped. It was felt, therefore, that whatever the results of experiments previously reported may have been, the methods employed completely vitiated the accuracy of the results. A technic was developed whereby the wall of the cochlea is drilled through with a no. 80 drill, care being taken to preserve a thin shell of bone at the base of the hole. If fluid escapes, the experiment is abandoned. After the hole has been made, its base is burned by means of a high frequency apparatus, the damage to the intracochlear structures being definitely restricted in this way. The animals may be tested either immediately or at varying periods after the scorching. It has been found that following this procedure individual tones are lost, most commonly high tones but occasionally low ones. The location of the hole can, of course, be changed at will. When these animals are tested and the high, low or intermediate tones are lost, a diluted solution of formaldehyde is injected through the heart and the ears are sectioned. When the sections are studied, a definite type of lesion is found, which though somewhat diffuse indicates that the method is practical. Photomicrographs demonstrate coagulation of the spiral ligament at the point of scorching, destruction of the cells of the stria vascularis and the sulcus, an extreme effect on Reissner's membrane and, in some instances, destruction of the organ of Corti. Charts were made to demonstrate the presence of tone islands in these animals, complete loss of certain tones and their return at other frequencies. The heterodyne audiometer was used entirely in these experiments, and with it tones graduated to tenths of octaves can be produced. The longest period elapsing between operation and testing has been three days. Animals may, of course, be studied which have been kept for much longer periods.

In all experiments the greatest possible care has been taken to avoid the troublesome phenomenon of magnetic interference, and it is felt as a result of this care the method is accurate and above reproach. Following every test for transmission a test for possible magnetic interference is made.

May 17, 1933

MOTION PICTURE DEMONSTRATION

Tic of the Auricular Muscles. Dr. A. M. Ornsteen and Dr. Edward E. Sprenkel, Jenkintown, Pa.

The Living Cilia of the Nose. Dr. Samuel R. Skillern, Jr.

Facial Paralysis as a Beginning Symptom of Mastoiditis: Operation and Recovery. Dr. Henry Dintenfass.

The Diagnosis and Treatment of Pharyngeal Diverticulum by the Combined One-Stage Method. Dr. Louis H. Clerf.

Directory of Otolaryngologic Societies *

FOREIGN

COLLEGIUM OTO-RHINO-LARYNGOLOGICUM ARMICITIAE SACRUM

President: Professor Gunnar Holmgren, Karolinska Mediko Kirurgiska Institut, Stockholm, Sweden.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg, 143, Groningen, The Netherlands.
Place: Stockholm, Sweden. Time: September 2-5, 1934.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

Chairman: Dr. W. P. Wherry, 107 S. 17th St., Omaha.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Place: Cleveland. Time: June 11-15, 1934.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
President-Elect: Dr. J. M. Wheeler, 30 W. 59th St., New York.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: 1934.

AMERICAN BRONCHOSCOPIC SOCIETY

President: Dr. W. F. Zinn, Medical Arts Bldg., Baltimore.
Secretary: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.

AMERICAN LARYNGOLOGICAL ASSOCIATION

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Secretary: Dr. W. V. Mullin, 9204 Euclid Ave., Cleveland.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. J. W. Jervey, 101 Church St., Greenville, S. C.
Secretary: Dr. R. L. Loughran, Bridgewater, Conn.
Place: Charleston, S. C. Time: April 3-5, 1934.

SECTIONS:

Eastern.—Secretary: Dr. Vernon L. Bishop, Medical Arts Bldg., Rochester, N. Y.
Southern.—Secretary: Dr. Fletcher D. Woodward, P. O. Box, 1685, University, Va.
Middle.—Secretary: Dr. Frank J. Novak, Jr., 30 N. Michigan Ave., Chicago.
Mid-Western.—Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.
Western.—Secretary: Dr. Ralph F. Davis, Mayer Bldg., Portland, Ore.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. John R. Page, 127 E. 62nd St., New York.
Secretary: Dr. Thomas J. Harris, 104 E. Fortieth St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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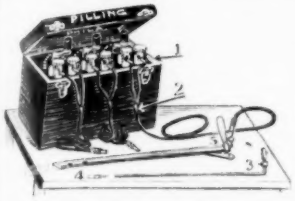

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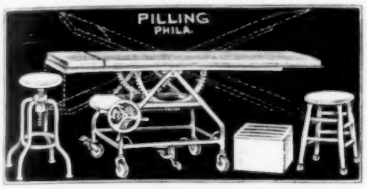

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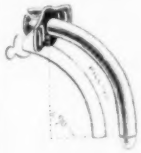

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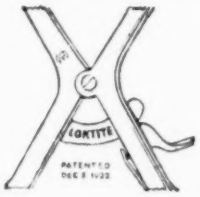

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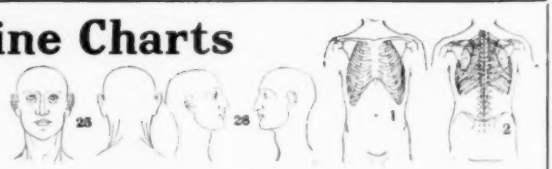

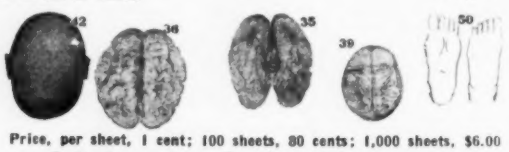

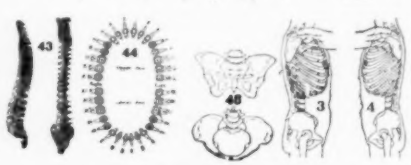

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